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An Address.[1]

By JOHN DAY,

Retiring President, Western Australian Branch of the British Medical Association.

SINCE the end of the War, tremendous changes have taken place in our way of life, and the winning of the War found us with greater problems than when the War began. We are living through a social revolution, when mankind is shifting its outlook, and the mere compulsion of tradition has lost its force. Since tradition has played such an important part in medicine, this social upheaval affects us deeply.

Science has advanced us into the atomic age; for good or evil, we have yet to see. Medical science has progressed at an unbelievable rate, and paradoxically has confounded us with unexpected problems.

One frequently hears the opinion voiced that these are terrible times to be alive, and who can show us any good that will come of them? Others say that this is a great time to be alive, and that the personal and public issues of it depend on whether we see that. What makes any era seem great or little to a man is the man's own eyes, his capacity of insight and vision. Human nature instinctively dislikes change, and we love to play safe by "staying put". We settle down in a familiar place, clinging even to its faults, rather than facing the unknown that alteration brings. There is in human kind a natural timidity that

> Makes us rather bear those ills we have
> Than fly to others that we know not of.

The three major forces of our time are fascism, communism and democracy. They stand in contrast, their unlikeness stressed and evident, but on one point they all agree. They are at one in demanding radical change, and we, as a democracy, are getting these changes. Thus in looking into the changes that are taking place in medicine, we must regard them only in their true perspective—as part of the whole change that is coming over our way of life.

Recently two members of our Association were idly discussing this and that, when the British Medical Association came into the picture. Said one: "The B.M.A.—I couldn't care less." That is the post-war saying, which is very familiar. We associate it with teenagers, or people on whom responsibility rests very lightly. The man was undoubtedly speaking his mind, but I would suggest that the indictment is of the man himself, rather than of the Association. However, it would be foolish to imagine that this attitude is confined to one member alone. The routine work of your Council goes on week after week, month

[1] Read at the annual meeting of the Western Australian Branch of the British Medical Association on March 27, 1953.

after month, but because nothing spectacular has happened it must not be said that nothing has been done. I have often had the feeling that your Council is looked upon by its members in much the same way as they look upon the Taxation Department—that it is an unpalatable necessity. I may be wrong in this view, it may be that I am unduly sensitive of what I consider our own inadequacy.

I have long felt that what is sadly lacking is criticism of your executive body. I would ask you to give this some thought. We badly need constructive criticism, and perhaps at times richly deserve criticism of the less palatable type. I would stipulate one condition: put it in writing. It must then come up for official consideration. Only through the interchange of views can we serve the Association and progress.

I address these remarks particularly to the younger members, because the future of medicine concerns you, much more than it does some of us, whose shadows, although we may not admit it, are already tilted towards the east.

Inflation of our monetary system is not the least serious of recent changes that the times have inflicted upon us.

The British Medical Association's experiences reflect that of every householder in the country, and every institution. This year in order to balance our budget it was necessary to raise the annual subscription, making it the highest in Australia. On the whole, this painful necessity was accepted very generously. Your Council is seriously concerned at the impact of rising administrative costs. The present form of administration, which has come into being during the last five years, is a comfortable one. But is it wasteful, is it inefficient, does it give us our money's worth? Many of the Council are not sure of the answers, and this will be one of their biggest problems during the coming year. I do not share the extreme view that there is waste and inefficiency, and that a total reorganization would halve our running costs. But it is difficult for the members, who are all busy men, to view this problem clearly and dispassionately and without prejudice. It may be necessary to follow the example of modern business practice, and to call in what is termed the business doctor to report on our office.

I think we can with justice be proud of our library and the service it gives, but we do not like the fact that it costs more to maintain than any other Branch library in Australia. We are indebted to our librarians, to the library committee, and particularly to the chairman of the committee because an immense amount of work has gone into this library. I regard the library and the post-graduate work as the greatest recent advances of our Branch. The cost of the library is heavy, but I do not consider it unreasonable. Many do not share this view, and I respect their views. The use and value of the library varies greatly between individuals, and a busy general practitioner, particularly the country one, cannot use its facilities like the consultants. However, it depends on your point of view. Do you ask: "What do I get out of the library?" or do you say: "What is its value to the Association as a whole?" It has been said that in maintaining the present library and its service we are living beyond our means. If this is the considered view of the majority, it may be necessary to curtail its service greatly. One idea that was explored was that of transferring the general office to the library site, which would result in considerable reduction in expenses. The hospital authority had no objection, but could not provide the necessary extra space.

Perhaps we may consider here for a moment the National Health Service of Great Britain. You will remember that the Beveridge Report of 1942 estimated an annual expenditure of £170,000,000 for the service, and this figure was accepted when the Act was brought into force in 1946. We know what happened. In 1949 Mr. Bevan, alarmed at the rising costs, issued instructions that the hospitals were to cut their costs by £9,000,000, but without curtailing the service. This is a typical ministerial edict. In spite of the Minister's exhortations, the reply was a bill of £400,000,000 in 1950. Costs had risen enormously between 1942 and 1949, but the planners failed to recognize this fact. Once a scheme gets well under way, it takes great political courage to amend and curtail it, and the exercise of such courage could well mean political suicide to the party in power.

It is well that we have this example of the National Health Service before us. If people are assured of free medical care for even the most trivial condition, the overall costs rise steeply. Our own Government has shown wisdom in introducing the scheme step by step. The "free medicine" was limited to the costly life-saving drugs and drugs of proven worth in the prevention of disease. The old age and invalid pensioners were then provided with a free general practitioner service. Now the next step of providing assistance towards the cost of medical care is about to be taken. The scheme is governed by sound insurance principles, and the patient is asked to pay part of the costs. I think the scheme is sound and may reasonably be expected to achieve what it sets out to do, without the crippling costs of the British scheme.

In the very near future this Earle Page scheme should really begin. Large numbers are already enrolled with benefit societies. When the Government scheme adds its contribution to the benefits paid for illness, it is hoped that the greater part of the population will join benefit societies.

Politically the scheme has one or two weaknesses. Firstly, it is claimed by the critics that it is a scheme for the healthy and not for the sick. The chronically ill cannot register with a benefit society for disease already known to exist at the time of joining. The fact that the Government will pay its share of the benefit on preexisting disease does not altogether answer the criticism.

Another criticism, receiving more emphasis lately, is that the main people to benefit will be the doctors. It is claimed that some doctors are raising their fees already to patients known to belong to benefit societies. This seems to apply particularly to operative procedures. Sir Earle Page has asked the Federal Council to appeal to all doctors to abstain from this practice. Any sudden or drastic raising of fees, either at this stage or during the early days of the new scheme, will defeat its main purposes and bring it into disrepute. The Federal Council asks us not to kill the goose that lays the golden egg.

Unfortunately, apparent instances of overcharging have been seized upon by the Labour Party. We understand that they are stating that they will fix the fees when they come to power.

Of course, there are many pros and cons to fixed fees, but developments over the past few years show that fixed fees would have great disadvantages. I think that at some general meeting our Federal representatives might be asked to give the inside story of various negotiations with Treasury officials, and of the latest appeal to the Federal Arbitrator by the repatriation medical officers. Any suggestion of Government fixation of fees should make us think earnestly of ways and means of discouraging those few members of the profession whose actions bring the whole of the profession into disrepute.

At this stage we might briefly consider the effects of our amazing progress in medical science. Some interesting facts come to light. Formerly only the fit survived. Now the not-so-well survive, but the incidence of sickness and the need for medical care are great in this group. Furthermore, the not-so-well hand on their weaknesses to their children, and you can imagine what a few generations might bring forth. On the other hand, many acute diseases, which formerly carried off the young and the fit, have been conquered. The increasing average age of the community has created many unforeseen problems. Old age is a degenerative condition and cannot be cured. But the aged by living longer have many more illnesses and need more and longer medical care. Thus the conquering of disease is like the winning of wars—victory brings new and greater problems to be solved.

And now I should like briefly to direct your attention to the most recent evil that has overtaken society. We always associate wars with great human suffering and death, and yet today road accidents are causing almost as much destruction as war. In the six years of the last war we lost 33,000 men. In the last six years 10,000 have died on Australian roads. In the war 180,000 were injured but not killed, and in the last six years 179,000 were injured but not killed on the roads.

We are creating a new section of the community, the civilian maimed and limbless, who will create a new economic burden and be the centre of much human suffering. Last night 80 beds at the Royal Perth Hospital were occupied by injured people and, as near as I can estimate, 65 of these were injured in road accidents. The implications are obvious.

If we had a sudden epidemic of poliomyelitis, and the Commissioner of Public Health said: "I have a new cure, I can save fifty lives, I can save two hundred people from being crippled", he would be hailed as a national hero. I believe that if motorists were educated and the *Traffic Act* enforced, we could save this number from death and injury. I think the whole community is waiting for a lead. They have long waited for the voice of authority, for the Premier, for the Minister of Police, for the Commissioner of Police to speak. But they have waited in vain. Have we used to the full every means at our disposal to combat the evil—the radio, the Press, the motion pictures? Of course we have not.

Last night in the Press the secretary of that emasculated body, the National Safety Council, estimated that the Easter toll of the road would be forty killed and injured. Watch the Press and see.

If there is one thing every government fears it is a surge of outraged public opinion. Do you not think that this Association should take the initiative and lead the people? Give it some thought.

In conclusion, I would issue two warnings for these changing times. Never face the future with eyes of fear, and never let us take ourselves too seriously.

FURTHER INVESTIGATIONS INTO ÆTIOLOGY OF GLOMERULONEPHRITIS.

By DORA BIALESTOCK,

Elizabeth Mary Sweet Fellow in Medicine, Department of Pathology, University of Melbourne.

IN the preliminary report (Bialestock, 1951) of this investigation into the ætiology of glomerulonephritis, the hypothesis that tissue sensitivity is significant in the ætiology of glomerulonephritis was discussed, and an experimental technique to explore this was described.

The preliminary results of tests on serum of patients with nephritis and without known kidney disease, respectively, showed that there was a statistically significant variation in the reaction of the sera of these two groups to normal human kidney extract. This was demonstrated by the collodion particle agglutination technique described in that report.

As a sequel, experiment was undertaken to see whether a difference between serum from patients with nephritis and those with non-nephritic kidney disease could be demonstrated.

As only human kidney extract had been used in the preliminary experiments, it was decided to investigate the question of the species specificity of the extract by comparing the material from horse, ox and pig kidneys with the human material.

Materials and Methods.

Serum was collected by myself from patients at the Alfred Hospital, the Children's Hospital, the Royal Melbourne Hospital and the Women's Hospital, Melbourne.

The method of collection and separation of the sera was the same as that previously described. As the number of sera handled in this series was far greater than in the previous investigation, the sera were preserved by deep-freezing at −23° F. Titres were not found to be affected significantly by this method of preservation.

The method of preparation of the kidney extract was the same as that previously described. The extracts from the horse, ox and pig kidneys were also prepared in the same way.

In the preparation of the collodion particles some difficulty was found because of variations in dispersion of the collodion when different batches of commercial non-flexible collodion were used. A mixture of the following composition was finally used in preparation of the particles and was found to give reasonably consistent results: nitrocellulose H.X. 8–13, 20 grammes; solvent (ethenol one part by volume to ether three parts by volume), 500 millilitres.

In the coating of the particles with the kidney extract, a slightly different technique from that previously described was used. This modification required a little less manipulation and did not affect the end-points obtained. To a concentrated suspension of fresh particles an equal volume of 5% kidney extract was added, and the mixture was allowed to stand in a water-bath at 37° F. for twelve hours. Double-distilled water was then added until a standard turbidity equal to tube IV on the Wellcome Turbidity Scale was reached.

Details of the actual testing were identical with those previously described. Control tests by the use of the test serum with uncoated particles, and coated particles *plus* saline were carried out on each serum tested. As a further check on the method, tests with strongly positive and low titre or "negative" sera were repeated with succeeding batches of tests. Whenever it was possible, serum from patients with nephritis was tested simultaneously with serum from patients with non-nephritic kidney disease.

Occasionally difficulty in reading end-points was found with opaque serum from some nephritic patients, or with other serum which was collected soon after a meal. Dilution of these sera prior to testing solved this problem.

Classification of Clinical Material.

The criterion of subdivision of the material was, of necessity, mainly on clinical grounds. In 15 cases, post-mortem material was available.

In the case of children and young adults, clinical subdivision was found to present few difficulties. It was in relation to the elderly patients, with vague histories and with moderately impaired to poor renal function, that difficulties in clinical subdivision were encountered. Fortunately, it was in these cases that the post-mortem material was often available, and it was in these that a mixed kidney lesion was usually present—as, for example, mild proliferative glomerulitis of the nephritic type associated with severe advanced arteriosclerotic changes or with chronic pyogenic infective changes. In these mixed cases, classification was made on the predominant lesion present.

In the clinical subdivision of nephritis a combination of at least three of any of the following features (in the absence of high fever, shivering, scalding on micturition and infected urine) were used in diagnosis: (i) preceding infective condition within three weeks of the present illness; (ii) hæmaturia; (iii) hypertension; (iv) increasing œdema in the absence of primary cardiac failure; (v) oliguria; (vi) albuminuria; (vii) granular and cellular casts in the urine.

A case was considered acute if the serum was collected within about four weeks of the onset of the renal symptoms.

Subacute cases included those in which there had been signs of "active" renal damage usually for several months.

TABLE I.
Titres of Sera Tested with Normal Human Kidney Extract.

Condition.	Degree or Type.	Titre.											Total.
		—	1·5	1·10	1·20	1·40	1·80	1·160	1·320	1·640	1·1280	1·2560	
Nephritis.	Acute	9	—	4	8	6	3	13	6	6	1	—	56
	Subacute.. ..	1	—	—	—	3	—	5	2	2	1	—	14
	Chronic and acute-on chronic ..	2	—	1	1	1	3	8	6	8	—	—	30
Renal disease other than nephritis.	Infective ..	9	—	1	3	1	0	3	—	—	—	—	17
	Toxæmic ..	4	2	2	4	2	2	1	—	—	—	—	17
	Malignant hypertensive ..	4	—	1	1	2	1	1	—	—	—	—	10
	Mixed group ..	14	—	1	2	5	—	1	2	—	—	—	25

Chronic cases were those in which there had been known renal damage following nephritis usually of several years' duration.

Cases of slowly progressive renal failure and those in which there was an acute exacerbation of chronic nephritis were included in this group.

In all there were 100 cases of nephritis, which were made up of 56 acute cases, 14 subacute cases and 30 of chronic nephritis and severe exacerbation of symptoms—popularly known as acute-on-chronic nephritis.

Other kidney diseases seldom presented difficulties in diagnosis. In the cases of pyelonephritis, a combination of dysuria with scalding, high fever, shivering, pyuria and culture of an organism from the urine was found. Included among the cases of infective renal lesions, the post-operative cases (usually after prostatectomy) also presented no difficulties. In all there was one group of 17 specimens of serum from subjects with infective renal disease.

TABLE II.
Reaction of Sera from Subjects of Nephritis to Kidney Extract from Several Species.

Number of Serum.	Species.			
	Human.	Horse.	Ox.	Pig.
1	7	—	2	—
2	—	—	—	—
3	6	2	6	1
4	5	—	4	—
5	7	4	5	2
6	5	2	1	2
7	6	1	3	—
8	5	—	3	1
9	4	—	1	1
10	6	2	—	—
11	6	2	4	1
12	3	—	—	—
13	6	—	1	2
14	5	1	—	2
15	7	—	—	2
16	7	—	3	—
17	6	—	2	3
18	7	1	—	3
19	8	—	3	2
20	6	—	2	—
21	5	—	1	2

The cases of malignant hypertension (10 cases) were all classical, as were the 17 cases of pregnancy toxæmia.

Other conditions included in the non-nephritic kidney disease were three examples of diabetes with albuminuria and single cases of myxœdema, subacute bacterial endocarditis, inferior *vena cava* thrombosis, septicæma, rheumatic fever, dental abscess, chronic osseous tuberculosis, tuberculous peritonitis, hydronephrosis, renal calculus, carcinoma of the kidney and carcinoma of the lung; in all these there was evidence of renal damage varying from transient albuminuria or hæmaturia with impaired renal function to frank uræmia with anuria.

Amongst the cases of acute nephritis were included 13 cases of the nephritis epidemic following streptococcal tonsillitis which was reported by Mansen and Wilson (1952).

In the carrying out of tests for species specificity of the kidney extract, fresh kidneys were obtained from animals immediately after death, and the fresh material was placed in normal saline prior to extraction by the method described fully in the preliminary report. Horse kidney was obtained from the Melbourne Zoological Gardens, and the pig and ox kidney from the City of Melbourne abattoirs.

The kidney extract from the horse and ox was darker in colour than the pig and human material. The consistency of the extracts was that of a flaky powder after freeze-drying. They all gave very strong Molisch reactions for polysaccharides and weak Biuret reactions for protein.

The tests with these extracts were carried out as for the human material alone. In all, 21 high titre human sera were examined and tests with extracts from all four species —horse, ox, pig and human—were always performed simultaneously on any one serum. The usual control tests mentioned above for each serum were always carried out.

Results.

In all there were 100 sera from patients with nephritis and 69 sera from patients with non-nephritic kidney disease.

Completely negative results were obtained in 12 of the nephritis sera and in 31 of the control sera. That is, there were 12% negative results with nephritis sera and 43% negative results with control sera. These results are presented graphically in Figure I.

Figure II represents the titre distribution in all the positive results obtained with nephritis serum, and in those obtained with serum from patients with other renal disease.

The average titre in the nephritis group is 1:160 serum dilution, as compared with 1:20 average serum dilution in the non-nephritic kidney disease group.

Table I represents the results obtained with all the sera tested. The number under each dilution column represents the total number of sera giving an end-point at that dilution for each of the groups tabulated.

A statistical analysis performed on these results by Dr. F. E. Bennett, of the Department of Statistics, University of Melbourne, yielded the following conclusions:

1. If all the "negative" cases are ignored and the "positive" cases only considered: (i) there is very strong evidence (by the F-test of Snedecor) that a positive reaction amongst nephritics at all stages is stronger on the

average than amongst any of the four non-nephritic groups; (ii) there is strong evidence (by the F-test) that the positive reaction amongst chronic nephritics is stronger than amongst acute nephritics; (iii) there is no evidence that the average strength of the four non-nephritic kidney disease groups shows any difference between the groups; (iv) although there is insufficient

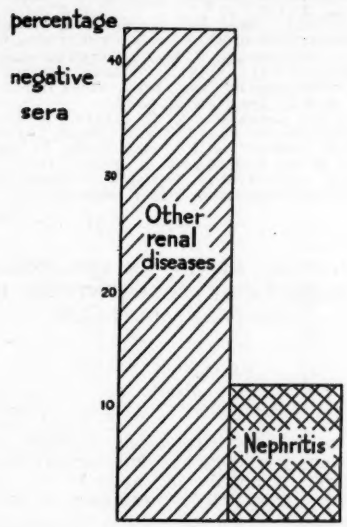

FIGURE I.

Diagram showing comparison of percentage of negative results obtained in nephritis and in other non-nephritic renal disease.

evidence to justify a definite statement (by the use of the F-test, linear regression), there is a rather more than strong suggestion in the data that the strength of the positive reaction amongst nephritics increases in this manner: acute, subacute, chronic.

2. If only the positive and negative results are considered and the strength of the reaction is ignored, the following conclusions can be drawn from the data given above: (i) There is very strong evidence, odds better than 1000:1 (by the use of the χ^2 test) that a positive reaction is more frequent amongst nephritis patients, taken as one class, than amongst the non-nephritic kidney disease patients taken as a single class. (ii) There is no evidence that there is any difference in the frequency of the occurrence of this serological reaction between the three stages of nephritis.

3. If the non-nephritic kidney disease groups are considered separately and all cases of nephritis are classed together, irrespective of the type: (i) There is strong evidence (by the χ^2 test), the odds being better than 100:1, that a positive reaction is more frequent amongst cases of nephritis than amongst cases of toxæmia of pregnancy, malignant hypertension and infective renal disease, when these three groups are taken together, and infective cases are taken separately. (ii) There is no evidence (by the χ^2 test) that the positive reaction occurs more frequently amongst cases of nephritis than amongst cases of toxæmia of pregnancy. (iii) Although the evidence (by the χ^2 test) is not strong enough to warrant a decision, there is a strong hint of the possibility that the positive reaction may be more frequent amongst nephritics than amongst malignant hypertensives.

The following table (Table II) will illustrate the strength of the reaction in 21 sera obtained when the extracts from horse, ox and pig were compared with the reaction to human kidney extract. For ease of representation the serum dilutions have been represented as the tube number at which the end-point occurred; for example, tube one equals a one-in-five serum dilution, tube two a one-in-ten serum dilution, tube three a one-in-twenty serum dilution, and so on up to tube ten.

The analysis of these results produced no evidence that the strength of the positive reaction of the kidney extracts from the different species could be correlated in any way.

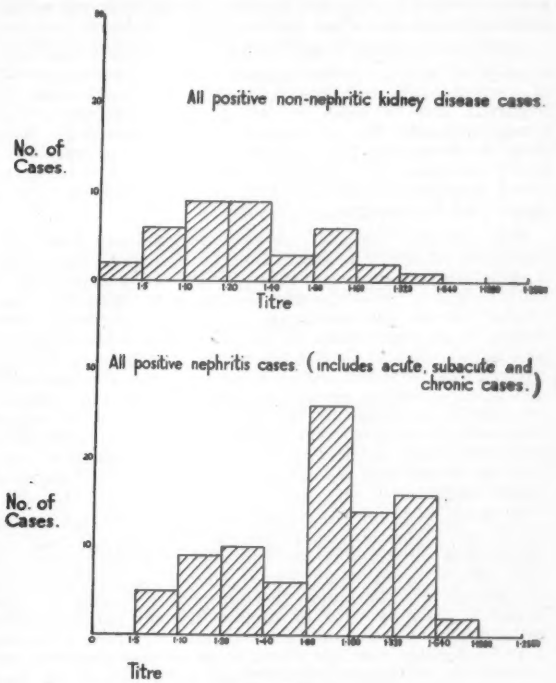

FIGURE II.

Diagram showing the titre distribution found in all "positive" sera in cases of nephritis and other renal disease.

Discussion.

In a comparison of the results obtained with those in the previous paper, it can be seen that a similar general pattern of titres has been found in both experiments, as far as the titre for the cases of nephritis is concerned. However, a striking difference is to be seen in the results in the control groups. Whereas in the controls with other kidney disease a scatter of titre is obtained (even though the average titres are lower than in nephritis) in the group of controls without known kidney disease, titres were never found in the higher serum dilutions. This is seen especially in the groups of pregnancy toxæmias—in which we are told that "there is no evidence (using the χ^2 test) that the positive reaction occurs more frequently amongst nephritics than amongst pregnancy toxæmias", and in the group of malignant hypertensives, in which we are told that "the evidence is not strong enough to warrant a decision that the positive reaction is more frequently found in cases of nephritis than in malignant hypertensives". Because of findings such as these, one must be extremely cautious in drawing any dogmatic conclusions as to the meaning of the results, even though there seems to be no doubt that both the strength of the serological reaction and its frequency of occurrence are greater in cases of nephritis than amongst combined groups of other types of renal disease.

The hypothesis that the ætiology of nephritis is in some way bound up with a hypersensitivity mechanism has been postulated since early in this century; but when a survey of experimental work to test this hypothesis is made,

conflicting results, such as those of Cavelti and Cavelti (1945) and of Humphrey (1948), may be found. However, although other explanations are possible, the bulk of data on animal nephritis would seem to support the hypothesis at least for these animal species used.

The very diversity of methods (Schwenkter and Complolier, 1939; Masugi, 1934; McLean *et alii*, 1951; More and Kobernick, 1951) used in the production of nephritis of the Bright's disease type, would favour the notion that kidney change is of significance in the development of the progressive disease when the stimulus is removed.

Apart from animal work, Lange *et alii* (1949) also used a collodion particle technique and a cell-free saline extract of normal adult and infant kidneys to test 23 cases of nephritis and 68 cases in which there was no known kidney disease. The pattern of results obtained by these workers (nephritis, 78% positive results and average titre 1:337, controls, 19% positive results and 1:78 average titre) conforms in general with results presented in this paper and the preliminary report.

The recently published paper by Glynn and Holborow (1952) is of great interest, in that it demonstrates that β-hæmolytic streptococci, the presence of which form a well-recognized predisposing condition to nephritis, can render human polysaccharide antigenic. Although few details of the experiment for the production of the nephritis and no histological details are given, it would appear that the use of a polysaccharide-kidney extract *plus* streptococci can produce lesions resembling nephritis. In the epidemic of nephritis described by Mansen and Wilson (1952), which was proved to have been associated with a streptococcal sore throat, the range of titres found in this group of cases differed in no way from that of the rest of the cases of acute nephritis.

Although it is not conclusive, the results of experimental work on nephritis would suggest that some sensitivity mechanism exists, whereby the polysaccharide moiety in the kidney is in some way rendered antigenic by the preceding infection, and the antibodies formed to this complex react *in vivo* to further normal polysaccharide with resultant local and general damage. That a similar type of damage can also occur in pregnancy toxæmia, and perhaps in malignant hypertension, must be kept in mind when a direct ætiological link-up is to be attempted.

Summary.

1. Results of tests on sera of 100 subjects of nephritis and of 69 subjects of non-nephritic kidney diseases, by the use of a predominantly polysaccharide human kidney extract, are given in detail.

2. The serum from nephritis subjects more often give a positive reaction and a higher titre of this reaction than does serum from the other subjects taken as a single group.

3. No conclusions pertaining to the ætiological significance of this reaction in nephritis should be drawn, as a similar reaction is found in some cases of toxæmias of pregnancy and in some of malignant hypertension.

4. No general pattern for species specificity of the kidney extract was found when horse, ox and pig extracts were compared with that of human.

Acknowledgements.

I should like to thank the medical and nursing staffs of the Alfred Hospital, the Children's Hospital, the Royal Melbourne Hospital and the Women's Hospital for allowing access to patients and for assistance in collection of specimens. I would express appreciation also to Dr. F. E. Bennett for his valuable work on the statistical analysis of the results.

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INFECTIVE HEPATITIS: AN ANALYSIS OF 1000 CONSECUTIVE NOTIFICATIONS IN WESTERN AUSTRALIA.

By D. J. R. SNOW,
Perth.

LITTLE has been written about infective hepatitis in Australia, and epidemiological data concerning it are difficult to obtain. The following analysis may therefore be appreciated by those who are interested in the disease.

Infective hepatitis was declared to be a notifiable disease in the State of Western Australia on January 9, 1948. Since that date, 1024 cases have been notified and 1000 of these notifications have been received since July 1, 1950. This number of cases occurring in a population of 591,602 (estimated as at December 31, 1951) constitutes an epidemic, which is still in progress. There is reason to believe that notification has been incomplete, so that notifications must be regarded as indicating minimal incidence only.

Epidemic hepatitis is a new experience in Western Australia. There are no official records of the prevalence of the disease in this State prior to 1948. The annual reports of the Commissioner of Public Health for some thirty years contain no reference to any outbreak of jaundice, and practitioners with long experience are unable to recall the occurrence of large numbers of cases over a short period of time. Groups of cases are known to have occurred within service establishments during both World Wars, but an epidemic involving the civilian population has apparently not occurred before.

Nomenclature.

In Western Australia the disease is notifiable as "infective hepatitis". This is in accordance with British nomenclature. American authors, however, prefer the term "infectious hepatitis", and in the International List of Causes of Death, "infectious hepatitis" is given precedence over "infective hepatitis".

An editorial article on the viruses of hepatitis in the *British Medical Journal* (1948) disposes of this question in the following terms:

> There can be no doubt about the nomenclature of infective hepatitis: this term was first used by Cockayne in 1912 and thus has priority over "infectious" or "epidemic", which was first used by Lindstedt in 1919.

A comment in *The Medical Officer* (1952), however, is of some interest in this connexion. It states that as early as 1907 the medical officer of health for Irthlingborough Urban District Council in Northamptonshire reported as follows:

> An unusual outbreak of what might have been called epidemic infectious jaundice occurred during the autumn, affecting a great many young children and causing one death. I apply the term "infectious" to the

condition, as in some instances several of, and in others all, children of a family were affected.

The difference in nomenclature may not be important, but it is associated with a subtle difference in connotation which is worthy of comment. The word "infectious" suggests a high degree of communicability from person to person, and such infections are usually spread by droplet and droplet nuclei. The word "infective", on the other hand, does not suggest a high degree of communicability, nor does it imply a method of spread similar to that of the common exanthemata. For these reasons the term "infective hepatitis" seems to be preferable, and has therefore been used in this article.

TABLE I.

Age Group. (Years.)	Males.	Females.	Total.	Percentage of Total.
0 to 4	20	12	32	3·2
5 to 9	118	144	262	26·2
10 to 14 ..	80	79	159	15·9
15 to 19 ..	51	55	106	10·6
20 to 24 ..	50	60	110	11·0
25 to 29 ..	52	42	94	9·4
30 to 34 ..	41	38	79	7·9
35 to 39 ..	30	21	51	5·1
40 to 44 ..	13	7	20	2·0
45 to 49 ..	8	7	15	1·5
50 and over ..	31	14	45	4·5
Not stated ..	14	13	27	2·7
Total	508	492	1000	100·0

Seasonal Distribution.

The chronological sequence of the notifications which provide the material for this analysis are indicated in Figure I, where the monthly notifications from January, 1948, to September, 1952, are set out. It will be seen that the epidemic began during the latter months of 1950 and reached a peak in June, 1951. Since then incidence has fluctuated, but has remained at epidemic level, despite the lapse of nearly two years. Notification levels are more conspicuous during the cooler months, and the general trend conforms to the English experience that "epidemics of infective hepatitis start in the autumn, rise to a maximum during the winter months, and subside in the spring" (Wilson, 1951).

Age Incidence.

The age distribution of notifications received is depicted in Figure II, which sets out the relative percentages of cases in succeeding five-year age groups. It will be observed that the five to nine year age group contributes the highest percentage (26%); 56% of all patients were aged under twenty years, and 76% under thirty. Two other features of interest are the relatively small proportion of cases notified in children below the age of five years, and the fact that 4·5% of patients were over the age of fifty years.

Sex Ratio.

The distribution by sex in the various age groups is set out in Table I, and it will be seen that the sex incidence is more or less equal. The male and female figures per 100,000 of each sex exposed are 166 and 171 respectively, which gives a male to female ratio of 1:1·03. This feature is of some interest, because the morbidity and mortality from communicable diseases are often higher in males, although there are some notable exceptions—for example, whooping-cough (Lancaster, 1952).

Mode of Spread.

Although a comprehensive field investigation has not been possible, inquiries have been pursued with the object of finding out whether any common factor such as food, water, milk or flies was concerned in dissemination. A number of homes in the metropolitan area have been visited and inspected, while patients or relatives have been interrogated. Results suggest that personal contact has been the main method of spread. A study of the behaviour of the disease during an institutional outbreak, which has been described elsewhere (Snow, 1953), and another of its pattern in large families, supports this view. Although contaminated water and other indirect methods of spread have been incriminated in a number of outbreaks, notably among service personnel, civilian epidemics have generally been attributed to spread by personal contact. The impression in this particular epidemic is that infection has occurred from person to person, and it is suspected that the mechanism is the transference of particulate fæces.

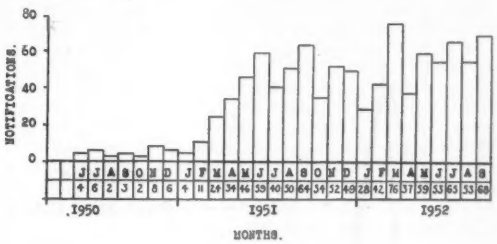

FIGURE I.

Hepatitis and Poliomyelitis.

There are a number of epidemiological and other similarities between hepatitis and poliomyelitis: both are virus diseases; both are characterized by spectacular clinical manifestations—jaundice in hepatitis, paralysis in poliomyelitis; while in both, a proportion of infections is non-icteric and non-paralytic, respectively. In both, droplet infection has been postulated as a mode of spread, although the evidence in favour of fæcal transference is more impressive. Both are preponderantly diseases of children and young people, and the opportunity is here sought of

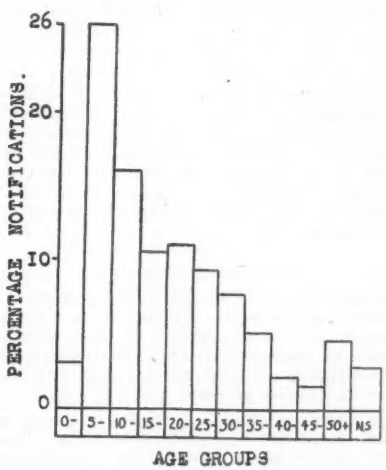

FIGURE II.

illustrating the comparison in the age selection of these two diseases in Western Australia. In Figure III, the relative percentage of cases in the various age groups for the two diseases is set out, and the similarity is striking. The only discernible difference is in the age group nought to four years, and the explanation here may well be that poliomyelitis, in its milder forms, is less difficult to diagnose in the very young than is a mild attack of hepatitis.

Public Health Aspects.

Segregation.

Viral hepatitis is a communicable disease. Segregation of the patient is therefore desirable. The balance of published evidence suggests that the intestinal-oral method is the main mode of spread. The duration of the presence of the virus in the fæces seems to be variable, but it would be wise to regard the patient's fæces as infective at least until he is completely free from all signs and symptoms. In epidemic times it is unavoidable that the vast majority of patients will have to be treated at home, and it should be an obligation on the family doctor to inform the mother of

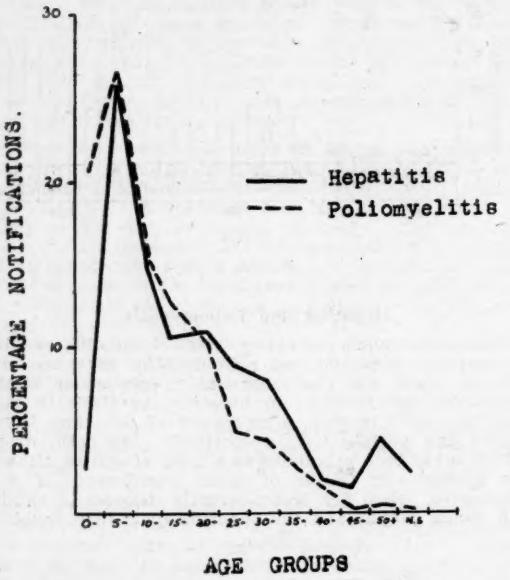

FIGURE III.

the possible modes of spread. When it is necessary to admit a patient to hospital, "barrier" precautions are advisable. In special circumstances isolation at a recognized hospital for infectious diseases should be considered. In Western Australia, children suffering from the disease and requiring admission to hospital are now admitted to the Subiaco Infectious Diseases Hospital rather than to the Princess Margaret Hospital.

Hospital Management.

Nurses need to be reminded that the patient's excreta are infective, that pyjamas and bed linen are likely to be contaminated, and that articles recently handled by the patient may be potential sources of infection. The fæces should be disposed of with special care and the bed-pan disinfected as soon as possible.

The importance of frequent hand-washing as a measure of personal prophylaxis should be stressed. Wash basins fitted with foot or elbow control are a distinct advantage.

The possibility of cross-infection through the agency of syringes, needles and lancets for capillary puncture should be borne in mind, and these should not be used for another patient until they have been sterilized.

Child Contacts.

The imposition of rule-of-thumb restrictions upon child contacts cannot be justified. The clinical attack rate of the disease is low and the incubation period is long. Exclusion of a contact from school should therefore be recommended only in the event of vomiting or other suggestive early signs.

General Measures.

Food, milk and water are all potential sources of spread of infection. The purity of these should therefore be safeguarded. Sanitary defects in private dwellings should be corrected, and flies must be suppressed. Finally, parents and school-teachers should be told that children must be constantly reminded to wash their hands after each visit to the toilet, and before each meal.

Blood Donors.

It is obviously undesirable to accept as blood donors persons from areas where hepatitis is specially prevalent. For this reason it is advisable that the local Red Cross Blood Transfusion Service should be furnished with a monthly list of notifications by the health authority. Appropriate adjustments can then be made in the blood-taking programme to avoid areas heavily affected.

The foregoing measures are unfortunately not specific. However, in the light of existing knowledge of the disease, they represent simple common-sense precautions, which appear to be both reasonable and desirable.

Summary.

A thousand consecutive notifications of infective hepatitis in Western Australia during the period from July, 1950, to September, 1952, have been analysed in regard to seasonal, age and sex incidence. It is suggested that the mode of spread during the epidemic has been by the transference of particulate fæces from person to person. Reference is made to some similarities between hepatitis and poliomyelitis. Control measures are briefly discussed.

Acknowledgements.

This contribution is published with the permission of Dr. Linley Henzell, Commissioner of Public Health in the State of Western Australia. In addition, I am indebted to Mr. J. F. Woolcott and Mr. A. C. Waldon for computations and diagrams, respectively.

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THE DISCOVERY OF WESTERN AUSTRALIA, WITH SOME EARLY MEDICAL HISTORY.[1]

By B. C. COHEN,
Perth.

AN oil refinery costing £40,000,000 is to be built at Kwinana and a cement works at Rockingham. The *Kwinana* and the *Rockingham* ran aground on the Success and Parmelia Banks in Cockburn Sound. Do the names mean anything to Western Australians? If only one could tune into a celestial wave-length, what would Thomas Peel be saying to Stirling? I imagine the burden of his remarks would be: "I told you so." For it was on the strength of Stirling's favourable report from the voyage of the *Success* that not only was Western Australia founded as a colony, but Thomas Peel was inspired with unbounded faith to launch his enterprise, his splendid folly, in attempting to settle the very area which is now destined to boom.

There are 500 members of the British Medical Association in Western Australia at present. Is it worth while recalling the incidents that led to the discovery of Western Australia and the experiences of our early colleagues?

[1] Read at a meeting of the Section of History of Medicine, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August 22 to 29, 1952.

Let us not imitate the English writer who, when he saw a memorial plate on which was written "John Lackland passed this way", said: "Well, he will not pass this way again, so let's get on to something fresh." Rather let us think with the American editor who relived the experiences of the characters in the books he read, who listened to the voice of the consuls when he saw a Roman coin, who heard the bleating of Abraham's flocks on opening his Old Testament, who lifted his Homer and shouted with Achilles. Hippocrates himself admitted that he learned something from the history of the Barbarians. So when you spend your next holiday at the Abrolhos, think of the wicked apothecary who carried on the doctoring for the good ship *Batavia* until he became a murdering pirate. Our Guildford colleagues no longer run the risk of being drowned whilst rowing a cow to Perth, as did poor Dr. Whatley, nor do they nonchalantly walk to York to visit a patient like their predecessor a century ago. Did you ever ride along the Balcatta road over the sawn-up tree trunks and know that they were called Hampton's cheeses after the shrewdest doctor that was ever in Western Australia, the only doctor-governor, who amongst other things provided the foundation of our town hall and brought the colony into a state of solvency?

In any historical review it is always difficult to fix a starting point. "Begin at the beginning", said the king in "Alice in Wonderland" gravely, "and go on till you come to the end, then stop." The initial stage of medicine in Western Australia (the *stadium incrementi*) is insidious, the *fastigium* or stage of full development has not yet been reached, the *stadium decrementi* or termination never will be. Nevertheless, for our purpose we may divide our life history up to the present time into various sections.

I have arbitrarily selected, firstly, the period from the first discoveries until the foundation of the colony in 1829; secondly, the period from the foundation until the commencement of the convict system in 1850; the next break I make at 1870, when a medical board was established to regulate the registration of doctors, seventeen in number, six of whom, although unqualified, secured registration because they had practised medicine before 1869. The fourth period is from 1870 until 1898, when the Western Australian Branch of the British Medical Association was formed with an initial membership of 23; and lastly, the period from then until 1952, when the membership was about 500.

In this paper I shall attempt to describe the first period.

Although a great number of references have been obtained with the assistance of Miss Lukis, of our Public Library, the actual medical side has been mostly gathered from the researches of one of Australia's best medical historians, the late Cyril Bryan, after whom our library is named. I have obtained most of his published and unpublished manuscripts, and with full acknowledgement have used scissors and paste on his writings wherever they fit in.

The discovery of Western Australia dates back to the sixteenth century during the voyage of Magellan (Fernao de Magalhaes) in his discovery of the Pacific in 1520. He was murdered by natives. There are pictures of Portuguese caravels, the ships of the period from 75 to 120 tons in which Magellan sailed, in the maps of Pierre Descaliers, 1550. Pierre Descaliers was a priest of Arques near Dieppe and a celebrated cartographer. In one of his maps, which bears the inscription "*Mappe Monde peinte sur parchemin par ordre de Henri II*" and hence called the "*Henri II Mappa Mundi* 1546", Java bears the name "*Java petite*", and below is the Australian continent called "*Java la grande*". There is further evidence that Western Australia was known to Portuguese pioneers of the sixteenth century in Cornelis Wytfleet's "*Descriptionis Ptolemaicæ Augmentum*", 1598, wherein, referring to *Terra Australis Incognita Magellanica*, he makes the following statement:

> Its shores are little known, the route deserted, the country seldom visited unless by sailors driven by storms. The Australis Terra begins at 2 or 3 degrees from the Equator, and it is maintained that if thoroughly explored would be regarded as a fifth part of the world.

There are some very interesting facts associated with familiar landmarks on our coast in connexion with the early Dutch explorers.

Dirk Hartog, in the ship *Eendragt* (1616), named the portion of Western Australia between Shark Bay and North West Cape "Eendraght Land" and put up his tin plate bearing an inscription. This was renewed in 1697 by Vlamingh, and after another century the two plates were picked up by the Frenchman Hamelin. He therefore called the spot Cape Inscription.

Dirk Hartog's Roads was subsequently named Shark Bay by the English medically minded Dampier.

In 1619 Frederick de Houtman, in the *Dordrecht*, discovered the reef which was named Frederick Houtman's Abrolhos. And when next you are catching fish and crayfish at the spot, remember that Abrolhos is derived from the Portuguese "*abri vossos olhos*" ("open your eyes"), for there are many dangers apart from broken beer bottles if you do not keep a sharp lookout. A member of that same expedition, Edel (after whom the adjacent land was called Edel's Land), with remarkable prescience, suggested to the Dutch East India Company that the land might bear gold.

In 1622 the captain of the Dutch vessel *Leeuwin* ("Lioness") rounded Cape Leeuwin and named the south coast (about Albany) Leeuwin's Land.

About this time a shipwrecked crew arrived at Batavia. It is of interest to note that the Dutch actually commissioned a boat (*Hazewind*) to search for the wreck and take possession of the land in the name of the United Province of Holland. For some reason the voyage was not made, otherwise the whole of Western Australia would have become a Dutch possession. The *Hazewind* met the returning *Mauritius*, and all put back to Batavia. It is very likely that the wreck occurred on the Montebello Islands, and may be located now that the atomic bomb research is taking place at that self-same spot.

In 1627 a very learned explorer, Nuyts, in the *Gulde Zeepaerd* ("Golden Seahorse") examined the south coast and called the land about the Bight Nuyt's Land.

The Honourable Pieter Nuyts was afterwards Ambassador to Japan and Governor of Formosa. He will be remembered by Western Australians every Christmas, when they see the brilliant orange-coloured Christmas tree called after him "*Nuytzia floribunda*". It was about this time that in the "Atlas" of Gerard Mercator (Gerhard Kremer) Hondius shows a small strip of land called Eendraghland.

In 1628 the tragic adventure of Pelsart was enacted. White settlers for the first time landed on Western Australian soil, and we find contact with a "medicine man". Certainly he was an apothecary; but until 1815, when the *Apothecaries Act* was passed, the borderline of those who practised the healing art was ill defined.

The medical apothecary was one Jerome Cornelis, who sailed as supercargo on the ship *Batavia*, commanded by Francis Pelsart. Cornelis had it planned to seize the *Batavia* and turn her into a pirate vessel. The *Batavia* was one of eleven ships, and in a storm became separated from the fleet and struck on one of the islands of Houtman's Abrolhos on the night of June 24, 1629.

W. B. Kimberley now describes the drama. Captain Pelsart, sick in his cabin, was roused by the impact (the ship in full sail). He saw the foam, which was actually breaking on the rocky islands, threw the cannon overboard and dropped anchor. Unfortunately a storm arose, making their position well-nigh hopeless. The women and sick were landed on one of the three nearby islands. The wine was broached by the sailors and their brutal behaviour rendered the situation more perilous. One hundred and eighty persons, some provisions and valuable merchandise were landed on the islands, but no water. They optimistically thought that fresh water was easily obtained in Western Australia. Heavy seas arose, and Pelsart was forced to "leave with the utmost grief his lieutenant and 70 men on the very point of perishing on board the vessel". To pacify the thirst-crazed shipwrecked members on shore,

Pelsart consented to take a boat and look for water. Then follows a harrowing description of their trip up the coast and their attempt to land on a barren, rocky shore with a heavy sea making it almost impossible. In places they swam ashore, battered by waves onto the rocks. Their description is familiar to all "Sandgropers". The barren country was without vegetation, sandy and level. A few natives were gathered about ashes and the remains of crayfish. Ant hills were observed so large as to resemble native huts, and there was a prodigious number of flies; but no water was obtained. Having searched for 400 miles, Pelsart courageously decided to make for Batavia, and with great skill and fortitude reached Java in three weeks. He immediately returned in the frigate *Sardam* to rescue the shipwrecked members of the *Batavia*. Then was exposed the turpitude of the medicine man. After Pelsart left, Cornelis proceeded to implement his previously conceived plan of seizing the boat for pirate purposes. Cornelis assumed command of the party and gathered round him some fellow traitors. They thereupon murdered the rest of the folk on their island, with the exception of a few men who swam to a neighbouring island, seven children and six women. Cornelis took a married woman for himself, presented the minister's daughter to his chief favourite, and held the rest for public use. The conspirators divided up the merchandise and jewels of the *Batavia*.

They next attempted by treachery to murder the occupants of the neighbouring island. However, these, under a member of the crew, one Weybehays, repulsed the murderers and took Cornelis prisoner. When Pelsart returned he was apprised in time and was able to overpower the would-be pirates. The ringleaders were executed, Cornelis had his hand chopped off and was hanged, and two conspirators were marooned on the mainland. As far as is known, they were the two first white inhabitants of Australia. Thus was the advent of medicine and settlement in the west a very grim one.

Reproductions in the *East Indies Journal* depict the following incidents of the voyage of Francoy Pelsart, of Antwerp, October, 1628: (i) the wreck and disembarkation; (ii) Pelsart sailing away; (iii) the massacre, showing a few tents and the fight in progress on a small island; (iv) the torture and execution of the mutineers—cutting off limbs and stringing up on gibbets.

The next Dutch explorers hold great interest for Western Australians, but not much from a medical point of view.

The great Tasman, who named Van Diemen's Land after the Governor of Batavia, explored our shores in 1644, and it is to him we owe the name of New Holland.

In 1696, Vlamingh, in the *Geelvinck*, whilst searching for the wrecked *Riddershap*, anchored off Rotte Nest (named on account of the rats and wallabies, whose native name is *quokka*) and then landed about Cottesloe beach. The party marched eastwards and came across a freshwater basin, now Fresh Water Bay. Further on they saw native huts "of a worse description than those of the Hottentots". I have not seen any Hottentots' tents, but from the knowledge of *mia mias* I can believe the description. They came upon a rare sight of the fabulous black swans described by Juvenal, so numerous that "our boat knocked over nine or ten". In his satires Juvenal gives the following description: "*Rara avis in terris, nigro que simillima cygno.*" ("A rare bird on the earth, and very like a black swan.")

They saw innumerable varieties of birds and "also heard the song of the nightingale", but could not meet the shy, retiring natives. Times have changed. I have never heard of a black swan being knocked over by a boat, nor of a shy retiring native; the varieties of birds on the river are shags, and the song of the nightingale is the laugh of the kookaburra. They named the river the Black Swan. They sailed north and departed, firing a cannon "as a signal of farewell to the miserable South Land". They took away Dirk Hartog's plate and left a pewter plate of their own, which was afterwards (1819) taken to Paris by Freycinet. The doctor on the voyage with Vlamingh was Victor Victorsz, overseer of the infirmary called the *Cranckbesoecker*.

About this time the English took an interest in Australian exploration. William Dampier, of Somerset, was the first Englishman to land in Western Australia. It happened that on a buccaneering voyage around the world he landed on the north-west coast of Western Australia, luckily at a suitable spot, which he named Cygnet Bay after the ship. He wrote the first authentic account of the country and the natives. Altogether he spent two months in the vicinity of Derby on this occasion. He wrote a full description of the natives, who made a very unfavourable impression on him. After leaving Western Australia the pirates quarrelled amongst themselves, and Dampier and some others were marooned on the Nicobar Islands. After many misfortunes he reached England, and his publication in 1698 so impressed the Admiralty that he was commissioned by William III to reexplore New Holland in the *Roebuck*. He arrived in Dirk Hartog's Roads, and renamed this place Shark Bay, from the prevalence of those fish.

Dampier was a man of many parts, explorer, pirate, naturalist, a keen observer, and for our theme an unqualified doctor. He wrote as follows, whilst at Tres Marias Islands:

> I had been a long time sick of a dropsy, a distemper whereof many of our men died, so I was laid down and covered all but my head in the hot sand. I endured it nearly half an hour, and then was taken out. I sweated exceedingly while I was in the sand, and I believe it did me good for I grew well soon after.

Later, when he was turned adrift by his fellow pirates, he wrote:

> I found my fever to increase, and my head so distempered that I could scarce stand, therefore I whetted and sharpened my pen-knife in order to let myself bleed, but I could not for my pen-knife was too blunt.

He described "the land animals which were a sort of raccoon differing from those of the West Indies, for these have very short fore legs and go by jumping about"—the first description of our kangaroo, and he gave a detailed picture of our bob-tailed lizard, "a sort of guana with a head at both ends, although the one end had no eyes nor mouth and the legs seemed to be all four of them fore legs seeming by the joints and by the bending as if they were to go indifferently either head or tail foremost".

He made the observation, which has been corroborated since, that this was a land of sand and flies.

He described with pictures quite a zoology—the red oyster-catcher with bright red bill and legs, the noddy of New Holland, the dolphin, as his men usually called the fish *Coryphæna hippuris* Linné, and the fish the seamen called the old wife—the picture looks something like that of a John Dory.

His descriptions discouraged further English enterprises in this direction until the famous voyage of Captain Cook. However, there was one to whom these stories appealed. This was Dr. Gulliver, a reputed cousin of William Dampier, whose travels were told by Jonathan Swift in 1726. Why this is worth recounting is because one of his adventures, that amongst the houyhnhnms, allegedly occurred on a small island off Albany (although according to the map it is possible that it was confused with Tasmania).

Let me recount the history of Lemuel Gulliver, who first and foremost was a physician and surgeon. He went to Emmanuel College in Cambridge and was apprenticed to a London surgeon, James Bates. He pursued his studies at Leyden, and for three and a half years sailed the seas as a ship's surgeon. He married and tried private practice, but gave it up because, as he sorrowfully said, "My conscience would not suffer me to imitate the bad practice of too many among my brethren". He eventually shipped aboard the *Antelope* in 1699 as ship's surgeon and headed for the East Indies. He was shipwrecked on what was described as Van Diemen's Land, and when, as sole survivor, he fell asleep on the shore, he awoke, as we all know, thanks to Dean Swift, overrun by the Lilliputians. According to the location given by Gulliver, "By an observation we found ourselves in the latitude 30 degrees 2 minutes south", he must have been half-way between Perth and Geraldton.

Swift probably had in mind Tasman's description and map, and was thinking of Tasmania.

Gulliver again made contact with Western Australia. On this occasion, in 1710, he sailed in the *Adventure*, combining the duties of captain and doctor. Before this, of course, he had encountered the Brobdingnagians, and there is one passage in the story that has a familiar ring to all Western Australians. It is a sentence that the late beloved James Mitchell was wont to quote:

> And he gave it for his opinion that whoever could make two ears of corn or two blades of grass to grow where only one grew before would deserve better of mankind, and do more essential service to his Country than the whole race of Politicians put together.

The adventure began very badly, with several of the crew dying of "calentures", and Gulliver recruited men from the Barbadoes. These turned out to be pirates, and promptly marooned Gulliver on an island. This island, according to his description, was just south of Albany or Esperance. Here he met the intelligent horse-shaped beings with the name only a "Quiz Kid" can spell — the houyhnhnms, who considered him only a yahoo. He finally left them in a canoe and sailed for what was the site of Albany. Here the natives attacked him and shot an arrow into his knee. He writes:

> I apprehended the arrow might be poisoned and paddling out of reach I made a shift to suck the wound and dress it as well as I could.

A difficult manœuvre to suck one's own knee. He was picked up by a passing ship and taken to London, and nobody except Jonathan Swift believed the story.

In 1791 the English Government sent Captain George Vancouver in the *Discovery*, and with him Captain Broughton in the *Chatham*, to sail to America via Africa and Australia. They entered the harbour of Albany and named it King George III Sound (the "III" was subsequently omitted). They named many of the places and took possession of the south coast in the name of the King.

There is much of medical interest in this voyage. Vancouver had been a midshipman to Captain Cook, that great man who applied the principles whereby scurvy disappeared from the list of killers. (It matters little whether he or Dr. McBride actually discovered the cure.) Cook's voyage lasted three years with one fatality; Vancouver's lasted five years with only one death from disease. Two surgeons and three surgeons' mates shipped on these boats. They carried "quantities of Dr. James' Powders, Vitriolic Elixir, oranges and lemons in such quantities as the Surgeons thought fit, together with a hundredweight of the best Peruvian Bark".

Alexander Menzies was one of the surgeons appointed at £80 a year by Sir Joseph Banks, but his duties were to be more of a naturalist than of a doctor, for he was instructed "to study the climate, report on the fertility of the soil, pack up samples of seeds, plants and shrubs, examine brooks and rivers, and submit earth and rocks to his microscope. The same with animals, birds and fish. To study the sea otter and sheep and bring back skins. To make friends with natives and study their customs, and attend their horrid repasts". I suppose he read novels in his spare time. Apparently he was satisfied with the arrangements, for he wrote to Banks rejoicing that the expedition was to call on the west side of New Holland, and that he would have a fine field for botanizing. Menzies went ashore at what is now Albany. The other surgeon, Cranston, was invalided to New South Wales.

We now must interrupt the English discoveries, for the voyages of the next English visitors, Flinders and King, were overlapped by a number of French expeditions, and they are replete with medical interest.

These expeditions were sent out by the French Government for the purpose of solving the mystery of La Pérouse's disappearance in 1785. In 1792 D'Entrecasteaux, commanding the *Recherche* and *Espérance*, sailed down the Western Australian coast. They named Recherche Archipelago, Cape D'Entrecasteaux and Esperance Bay. The zoologist Riche was lost in the bush near Albany, but ultimately made his way back after fifty-four hours without food.

In 1801 a further expedition was sent, as the La Pérouse mystery was still unsolved. Baudin was in charge of the *Géographe*, and Hamelin commanded the *Naturaliste*. Having named Cape Naturaliste and Geographe Bay, they encountered a severe storm, and although Baudin had named Rottnest for their rendezvous, he sailed on to Shark Bay, and after making investigations there he departed on to Timor without waiting for Hamelin. All the records show Baudin as a man of very unpleasant character.

After the storm had subsided in Geographe Bay, Hamelin carried out his instructions and made for Rottnest. He explored the islands south of Rottnest, which he named Berthollet and Buache Islands; these were afterwards changed to Carnac and Garden.

A party under Heirrisson explored the Swan River and found the islands on which the causeway is very slowly being rebuilt.

Baudin and Freycinet returned in 1803, and Freycinet again in 1818. No further explorations were undertaken by the French Government, but in 1826 Dumont D'Urville commanded the *Astrolabe* for purposes of scientific research.

It was partly due to the unfounded fear that the French had designs on Western Australia that Governor Darling urged the Home Government to hasten the founding of our colony.

Now there were many famous French doctors on the voyages just mentioned. Owing to the indefatigable work of Cyril Bryan there is a letter from the Minister of the Navy, Paris, giving a detailed list of all the French doctors associated with the explorations. Keraudren did not accompany the expeditions, but issued instructions to all aboard Baudin's voyage. Short accounts will now be given of some of them.

Dr. L'Haridou gained his first-class diploma for a thesis entitled *"Des affections tristes de l'âme considérées comme causes essentielles du scorbut"*. That would be regarded now as ultra-psychosomatic—sad affections of the soul considered as essential causes of scurvy. L'Haridou deserves a special mention for his wonderful work on the voyage with Baudin. Of his skill and his character Péron cannot speak highly enough. He courageously combated a deadly snake bite, he attended his ungrateful commander uncomplainingly, and he sold his private possessions in order to buy medicine, food and even water when Baudin refused to help, although the number of dead and dying in the crew was increasing. Remember L'Haridou when next you visit Shark Bay. A quiet, peaceful basin of water behind Peron Peninsula is named L'Haridou Bight after that peaceful, gentle and skilful doctor.

Dr. Bellefin also wrote concerning scurvy, and gave directions for the use of drugs for sea captains.

The famous Dr. Taillefer was admitted as an "extern" to the *Hôtel-Dieu* of Paris in 1795. He was taken prisoner of war in England, but contrived to escape. He went on the Australian voyage of discovery on the *Géographe* in 1803, and was made a doctor of medicine. He worked his way up, received the Legion of Honour Cross from the hands of Napoleon, and was named Officer of the Legion on the battlefield of Montmirail. He was the surgeon-in-chief of the naval forces. He fought in Spain, Prussia, Poland, Pomerania, Austria and Germany. He returned to an honourable civil practice and died at the age of eighty-seven years. He wrote articles "Concerning Dysentery and Cholera", "Prevention against Plague", and "About the Properties of Corrosive Sublimate in Wood Preserving and its Industrial Hazards".

On the same expedition were Dr. Péron, Dr. Lesueur and Dr. Leschenault.

Péron is a name very familiar to Western Australians. Point Peron will be still more familiar when the Kwinana area is exploited. Francis Péron at an early age was drafted into the French army. A German bayonet which tore out his left eye ended his soldiering and he returned

to Paris to graduate as a doctor. He was rejected by his lady love, which indicates that she was just as one-eyed as Péron, and he turned his affections to zoology. He was a great historian and naturalist to Baudin's expedition, and his journal describes the criminal foolishness of Baudin, which materially helped in losing Péron's scientific comrades. There is a wonderful obituary eulogy written by the chief medical officer Keraudren on the occasion of Péron's death in 1810, at the age of thirty-seven years, from a chest complaint.

Nothing could restrain the young doctor from making the arduous voyage in search of new scientific zoological facts. M. Cuvier stated that Péron and Lesueur collected more than 100,000 new specimens, more than the total collected by Cook, Carteret, Wallis, Furneaux, Meares and Vancouver all put together.

Péron wrote books on physics, medicine, political economy and natural history. Unfortunately he was able to complete only one and a half volumes of his history and atlas.

The obituary notice is a lavish eulogy. It was a charming thought for Lesueur to design for the monument a model of the ship *Géographe*, under which he put the following inscription:

> *"Il avait de grands talents et cependant il eut beaucoup d'amis."*
> (He had great talents and yet many friends.)
> *"Il s'est désséché comme un arbre chargé des plus beaux fruits qui soccombe a l'excès de sa fécondite."*
> (He withered away like a tree laden with the finest fruits which is killed by its own excessive fruitfulness.)

On board the *Recherche* and *Espérance* were Dr. Renard, Dr. Boisdeliot, Dr. Joanet, Dr. Goffre and Dr. Labillardière, who published a work on Australian flora.

With Freycinet were Dr. Quoy, Dr. Gaudichaud and Dr. Gaymaud. These, together with Lesson and Hombron, all carried out valuable investigations, but I have not included details, as they are more of Australian than of Western Australian interest, and because they approach the end of the period of exploration and overlap the time of the founding of the colony.

At this stage we must revert to the English explorers Flinders (1801) and King (1817).

Flinders anchored in King George III Sound and surveyed the immediate coast. He compiled a short aboriginal vocabulary and made notes of the habits of the aborigines. He was sailing the coast simultaneously with Baudin. We owe the name Australia to Flinders. Previously the land was known as Magellanica, Java La Grande, Terra Australis, New Holland, and part as New South Wales. In 1814 Flinders's suggestion of Australia for the whole continent was adopted.

Phillip Parker King made several voyages on which Dr. Hunter accompanied him. Charts made by them are still used.

Flinders was the son of a Lincolnshire doctor, grandson and great-grandson of doctors. He abandoned medicine for the call of the sea, mainly because of reading the story of "Robinson Crusoe". It was our own William Dampier who was instrumental in marooning Alexander Selkirk on the island of Juan Fernandez, and incidentally another doctor comes into that piratical story—Dr. Dover, famous for Dover's powder.

On one occasion at King George Sound Flinders's life was saved from attacks of aborigines by the gallant action of Dr. Purdie, one of the ship's doctors, who courageously went up unarmed to meet the natives.

We are doubly indebted to the French explorers, because not only did they perform yeoman service in the discovery of our country, but they gave rise to fears that the western and southern parts of Australia might eventually be annexed by the French Government. Therefore General Sir Ralph Darling, of New South Wales, urged the Home Government to take steps to avert this. He first of all sent Major Lockyer with a detachment of the 39th Regiment and Captain Wakefield with some convicts to settle at King George Sound, and in the next year Captain James Stirling in His Majesty's ship *Success* accompanied by botanist Charles Fraser. Lieutenant Carnac was on board, and his name was given to Berthollet Island (now Carnac Island). Mr. Fraser ascended the hills which he named the Darling Ranges.

Captain Stirling reported so favourably that the Home Government acted on his recommendations, and he was personally entrusted with the charge of organizing the expedition for the colonization of Western Australia.

There were many doctors who accompanied the *Success*, the *Parmelia* (Captain Stirling) and the *Challenger* (Captain Fremantle); but I have now given a brief survey of the explorers and the doctors of the period up to the expedition of colonization, and the next period from then to the coming of the convicts I must leave until a future occasion.

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ESTABLISHING BREAST FEEDING.

By ALAN CHERRY,
Largs Bay, South Australia.

Cows feed their calves and primitive women breast feed their babies. In a civilized community this is far from true. Why? Do most women wish to breast feed their children? Yes. *Primiparæ* almost invariably wish to do so, and the *multipara* who has failed before would if she could. Why cannot they all feed their babies?

The simplest way to tackle the problem is to consider why the *multipara* failed to feed her previous children. The reason may be any of the following: (i) she had no desire to breast feed the baby; (ii) the child would not take the nipple; (iii) she had sore nipples or breasts with or without abscess formation; (iv) there was insufficient breast milk.

1. The first reason is more common than the mother wishes to admit. It is usually possible to assess this during the pre-confinement attendances, and if the practitioner is convinced that the mother is determined not to "mess around with breast feeding", then he had best establish the infant on the bottle from the start.

2. Leaving out deformities of the nipple, the breast or the child, I think that the cause here is that the baby quickly learns that it gets something from the spoon or bottle; it just cannot be bothered with the nipple.

3. Sore nipples *et cetera* present a difficult problem. For the cracked or fissured nipple penicillin cream is desirable, but for the tender nipple pile ointment (for example, "Hæmorrhex" or "Renaglandin") is on its own. Sore nipples can dry up the milk supply, as the mother dreads the next feeding and hates the approach of her child and secretly cannot wait for it to be put on the bottle. I am now preparing the nipple before confinement with pile ointment in place of lanoline.

4. Insufficiency of breast milk is easily the most common category. Most Australian children are born in hospitals. Hospitals must have a system for efficient working. Trainee nurses must be taught a system; unfortunately the wicked baby rarely conforms to any system, and if it does not, then of course the system is not wrong. If mother and child are fortunate enough to conform to the hospital's system they leave with breast feeding fully instituted.

The principles of the average system are as follows: (i) "The mother must have a good rest; after all, it is the only one she ever gets." (ii) The baby must be taught regular habits and discipline from the start (that is,

feeding times are determined by the alarm clock rather than by appetite). (iii) Adults rest their stomachs at night, therefore the child must get indigestion if allowed to gorge at will. (iv) The child must not be allowed to lose too much weight and must never leave hospital below birth weight. The child is therefore quickly introduced to the scales, and elaborate tables are kept of every feeding. These magnificent tables alone enable one to assess the child's progress.

In practice these principles work as follows.

From the first to the third day there is very little colostrum or milk, and the child is losing weight. Obviously it must have something—just an ounce or so of sweetened water, "Karilac" or borrowed breast milk. The child is taken to the breast every three or four hours, of course, but it does not suckle so hungrily as it would if there had been no supplementing or artificial feeding. In consequence early breast stimulation suffers.

On the third day the milk is beginning to flow, and each feeding can be checked with the famous standards. There are three categories: (i) The amount taken is almost the same as the standard—lucky child! (ii) The child gets less than the standard feeding, and is therefore put back to chew or play with the unproductive nipple, which becomes sore in the process, and the baby gets a belly full of wind. The child's feeding is still below standard and must obviously be supplemented. Supplementing reduces breast stimulation at the next feeding and gradually replaces breast feeding completely. (iii) The child gets more than the standard feeding. This is "overfeeding". Less time at the breast is allowed for subsequent feedings, and the child gets less total breast milk than it wants from 6 a.m. to 10 p.m., and therefore cries from hunger at night. It leaves hospital apparently established and contented on five breast feedings and a "silent" midnight nursery bottle. At home the baby howls all night. Mother has not heard about its bottle, does not weaken by giving it the breast, and nobody has any sleep. Mother and father are soon frantic and the bottle is on the way.

After the 10 p.m. feeding all the babies are herded into the nursery, where the obedient ones sleep through until 6 a.m. If a baby cries and is not satisfied with a clean napkin and a "bedmake", what are the night nurses' alternatives? (*a*) Let it cry and wake all the rest? (*b*) Take it to the mother for a breast feeding? The system says "no", and anyway it would disturb the other mothers. (*c*) Bottle feed the child with water or with artificial or borrowed milk? Obviously this is the correct solution and is very commonly used, in spite of fervent avowals to the contrary. This night artificial feeding further reduces breast stimulation. I sympathize with every nurse who does this—what else can she do under the present system?

These are some of the reasons why the *multipara* failed to feed her last child. The cause must be sought at her pre-confinement visits, the remedy must be explained, and then, if she is eager to give breast feeding another try, one must endeavour to make her confident that she will feed this child. Such a *multipara* and all *primiparæ* should be given some warning of the pitfalls that lie ahead, although care should be taken not to frighten the timid ones, and to make certain that the patients realize that these pitfalls have a ready solution.

What is the answer to this problem? Why not feed children naturally instead of having all this system? Because "the mother has other things to do besides fooling with her baby all day, and the child must be taught habit and routine in a high pressure civilized community". This is agreed. I consider that both the system and demand feeders are right, but at different times. Feeding should be on reasonable demand until the mother is completely confident that she has sufficient milk and can feed her child. Then, and only then, should discipline and habit training begin. At the earliest sign of feeding inadequacy demand feeding is recalled, and so breast feeding can be maintained until weaning is desired.

At this juncture I would like to pay tribute to the love, care and devotion shown by the nursing staffs of hospitals and child health centres, particularly the former. Most of what little knowledge I have of feeding problems has been gleaned from them; but, while admiring their enthusiasm I do disagree with many of their rigid beliefs. These nurses have rarely had children of their own, they rarely see the mother and baby in the home, and of course they are never present during the early morning hours of the first few days after homecoming. This makes a vital difference in our approach; the hospital staff thinks too much of the child's condition on its discharge from hospital, and the health centre sees the child for brief periods under totally artificial conditions.

I believe that maternity nurses should spend part of their training residing in the young babies' homes—at least for the first week after discharge from hospital.

I believe that the *régime* in hospital should be as natural, homely and undisciplined as is consistent with hygiene and privacy. The mothers should be encouraged to sit out of bed on the second day, with gradual and progressive ambulation. This early mobilization enables the mother to leave hospital feeling far fitter, fresher and more able to handle her domestic duties than does the prolonged stay in bed advocated in the past. Numerous mothers have testified to this—and this, to my mind, disposes of the first principle of the system—that is, the rest. This early ambulation enables the mothers to help themselves and each other more, and in this way babies requiring a night feed can either be taken to the mother or have mother brought to them.

I believe that mothers should be encouraged to help themselves more and do more for their babies, so that the nursing staff would be freed of some of their chores. This, coupled with the abolition of most of this "weighing racket", would lessen the demand for staff and thereby lessen the costs to the patient. By "weighing racket" I mean the incessant "before and after feeds" weighing. The child's temperature is not taken before and after feeds, nor is it taken at all unless such a precaution is indicated. Weighing is a method of checking on the abnormal, and routine weighing in the normal case serves merely to make the mother obsessed with the scales and panic-stricken at the slightest variation. To me, contentedness, wet napkins, normal motions and a healthy appearance are far more important than these scales. If these requirements are not met, then by all means have resort to the scales, thermometer, stethoscope *et cetera*.

How long should mother and baby remain in hospital? Ten days? Fourteen days? Some hospitals are still charging for the fourteen days whether the patient is there or not. This is far too systematized. I believe in sending mother and child home as soon as one is satisfied that the mother can cope with her baby and household too. In some cases this is after only two or three days; in others it may be two or three weeks. Here again, the sooner they go home, the smaller the bill; but it means extra visiting for the doctor—extra visits that are well worth while if they can solve early feeding problems before they become insuperable obstacles.

THE EFFECT IN VITRO OF SALICYLATES ON BLOOD SEDIMENTATION.

By G. A. W. JOHNSTON and H. M. WHYTE.
From the Kanematsu Memorial Institute of Pathology, Sydney Hospital, Sydney.

IN some diseases, such as the rheumatic disorders and tuberculosis, the erythrocyte sedimentation rate is taken as a measure of pathological activity, and serial estimations give an indication of progress and the effectiveness of treatment. However, when salicylates are included in the treatment there is some doubt as to whether the sedimentation rate is a reliable guide to progress, because it is reported that salicylates reduce sedimentation of their own accord without necessarily influencing the underlying disease process. It is of considerable importance to know

whether this is true, as salicylates of one type or another play an important part in the treatment of rheumatic fever and rheumatoid arthritis, and, as *p*-amino salicylate (PAS) they have more recently gained a prominent place in the treatment of tuberculosis.

The belief that salicylates inhibit sedimentation is based on the results of experiments made both *in vivo* and *in vitro*. Homburger (1946) and Rapoport and Guest (1946) found that the blood sedimentation rate could be reduced in patients suffering from rheumatic fever, lipoid nephrosis, osteomyelitis and carcinomatosis when aspirin or sodium salicylate was given by mouth. As it seemed unlikely that these drugs would be correcting the pathological processes in such a variety of diseases, they attributed the changes to some direct effect of salicylates on the factors which influence sedimentation. This belief was strengthened by reports (Bendien, Neuberg and Snapper, 1932; Lichty and Hooker, 1941; Homburger, 1945) that sedimentation could be inhibited by the addition of sodium salicylate to blood in the test tube. The mechanisms of these actions are unknown, and whether the same mechanism applies in both situations is uncertain. For this effect to be produced *in vitro*, either very high concentrations had to be used, much higher than are ever produced therapeutically in the blood-stream, or the blood had to be stored for twenty-four hours or so before ordinary therapeutic concentrations were effective.

It is our intention to investigate these problems, and here we present the results of those experiments *in vitro* which are most relevant to the clinical aspects of the topic. We have repeated and extended the work that has been done previously with sodium salicylate, and have examined in a similar way sodium *p*-amino salicylate, aspirin and salicylamide. Salicylamide is said to be less toxic than the commoner salicylates, but equally effective in the treatment of rheumatic diseases (Litter, Moreno and Donin, 1951).

Method.

Standard techniques for measuring sedimentation rates were used: the Westergren method with one part of 3·8% sodium citrate solution to four parts of blood, and the Wintrobe method with heparin or dry oxalate mixture as anticoagulant. Results were recorded as millimetres of sedimentation in the first hour.

To ten parts of the blood-anticoagulant mixture one part of physiological saline or a solution of salicylate in saline was added. The samples were mixed by being tipped a constant number of times before the sedimentation tubes were filled. Tests with "fresh" blood were made within thirty minutes of the drawing of the blood, and tests with "heated" blood about ten minutes after removal from a water bath maintained at 37° C. The concentrations of drugs used in these experiments were selected to extend well beyond the usual therapeutic plasma levels of 0 to 60 milligrammes *per centum* for sodium salicylate and 0 to 10 milligrammes *per centum* for PAS. The plasma concentrations mentioned in the results were calculated from the known amounts added to the blood, the hæmatocrit values and the assumption that practically no salicylate enters the cells (Lester, Lolli and Greenberg, 1946).

The patients whose blood was tested suffered from a variety of diseases, including rheumatoid arthritis, pulmonary tuberculosis, pneumonia and cancer. Each experiment was designed and conducted so as to permit of statistical analysis of the observations; results were regarded as significant only if the probability of their being due to chance was less than 1%. The accuracy of the methods was such that the standard deviation in any one experiment did not exceed 2·8 millimetres. The main effects described below have been reproduced with different anticoagulants and from patients with different diseases.

Results.

Sodium Salicylate.

The effects of sodium salicylate in fresh blood, in stagnant heated blood and in mixed heated blood are set out hereunder.

In Fresh Blood.—The presence of sodium salicylate in freshly drawn blood generally makes no difference to the sedimentation rate (Figure I). Slight effects are sometimes produced, as is evident in Figures II and III.

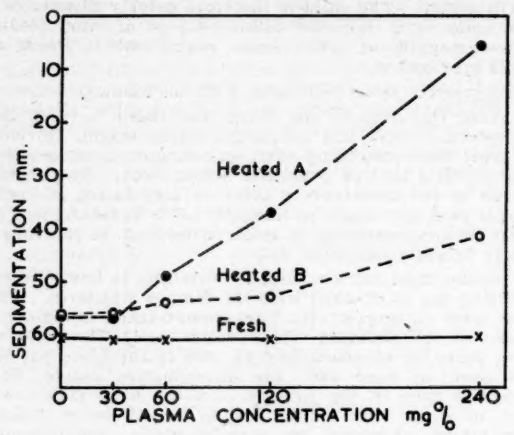

FIGURE I.

The influence of sodium salicylate on sedimentation of blood, fresh and when added before (B) or after (A) being heated at 37° C. for three hours (Wintrobe method, heparin).

In Stagnant Heated Blood.—If blood is allowed to stand for twenty-four hours at room temperature, its sedimentation rate is reduced. If salicylate is present a further reduction of rate becomes apparent. This unmasking of an inhibitory effect of salicylate can be hastened by keeping

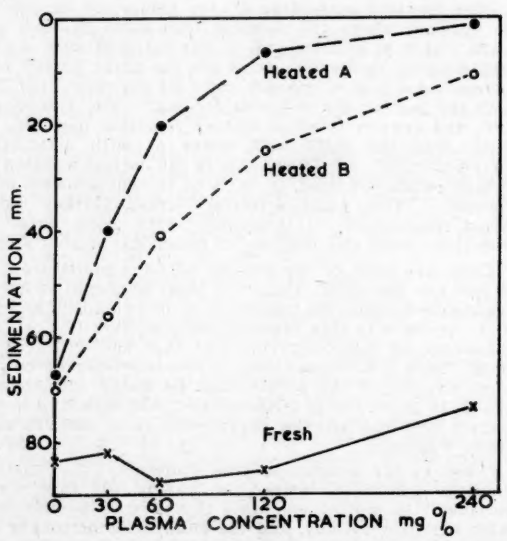

FIGURE II.

The effect of sodium salicylate, the Westergren method being used.

the blood at 37° C., and as a standard procedure we have done this by standing the tubes in a water bath for three hours. The results of one such experiment are shown in Figure I. It can be seen that the addition of salicylate after the period of heating produces an effect which is even greater than when the drug was incubated with the blood. It may be concluded that after standing and being heated

some change occurs in blood, making it susceptible to an action of sodium salicylate which results in a slowing of sedimentation.

In Mixed Heated Blood.—Fahraeus (1921) held that continuous mixing of blood during heating prevented the slowing of sedimentation caused by stagnation and heating. To confirm this and test the possible influence of mixing

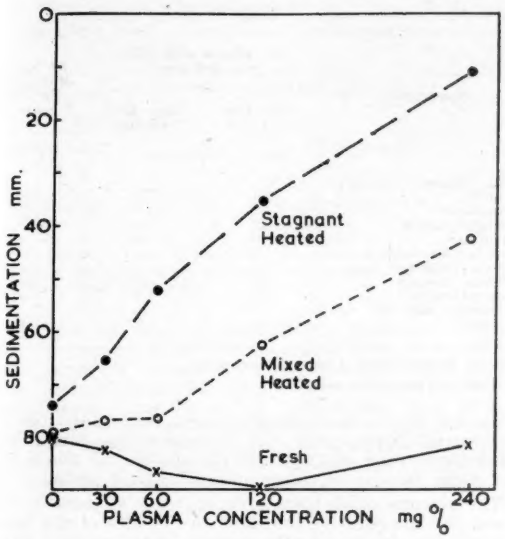

FIGURE III.

The effect of continuous mixing during heating on the inhibition of sedimentation caused by sodium salicylate (Westergren).

on the action of sodium salicylate, the samples of blood were put into ampoules which were sealed and mechanically rotated continuously in the water bath while other tubes of blood stood stagnant in the same bath. The sedimentation rates of these mixtures were then estimated; an example of the results is shown in Figure III. It can be seen that blood containing no salicylate formed a sedi-

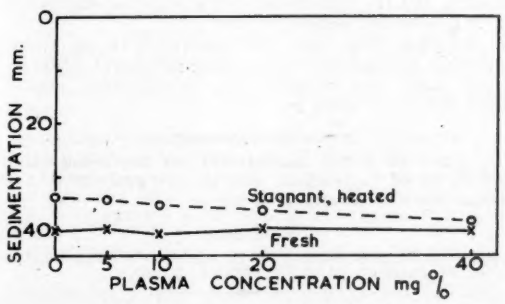

FIGURE IV.

The effect of sodium PAS on sedimentation (Westergren).

ment as readily after being mixed and heated as fresh blood, whereas blood which had been stagnant during heating did not form a sediment so rapidly. This confirms Fahraeus's observation. It can also be seen that the mixed blood was affected less than the stagnant blood by the salicylate. Indeed, salicylate concentrations of 30 and 60 milligrammes *per centum* had no significant effect on the mixed blood in this experiment.

Sodium p-amino Salicylate.

PAS, like sodium salicylate, caused little or no change in sedimentation rate in fresh blood. However, it acted in quite the opposite fashion to sodium salicylate in blood which had been allowed to stand, in that it caused an increase in sedimentation rate. Figure IV shows that heat and stagnation caused a reduction in the sedimentation of blood which contained no PAS, and that increasing concentrations of the drug progressively increased the sedimentation rate almost to that of the fresh blood. PAS, like sodium salicylate, was just as effective when it was added after the blood had been heated, while continuous mixing during the period of heating reduced its effect.

The effects of PAS, though more variable and much less striking than the sodium salicylate effects, were nevertheless statistically significant. Whereas PAS caused acceleration, sodium salicylate caused retardation of sedimentation; but neither drug exerted its effect unless blood had first been made susceptible by stagnation and heating.

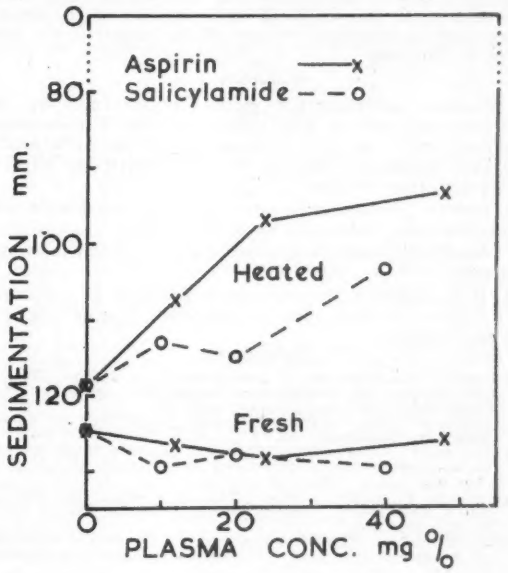

FIGURE V.

The effect of aspirin and salicylamide on sedimentation (Westergren).

Other Drugs.

Aspirin and salicylamide retard sedimentation in similar fashion to sodium salicylate (Figure V). The range of concentration of these drugs available for testing is limited by low solubility.

Discussion.

These results confirm reports that sodium salicylate can decrease the sedimentation rate of blood *in vitro*. Aspirin and salicylamide produce the same effect. On the other hand, PAS acts in quite the opposite fashion, in that it causes blood to form a sediment faster—a rather surprising result in view of the similarity in chemical structure of all these substances. Whether these effects *in vitro* are a true indication of what happens in the body when these substances are used in treatment is one aspect of the investigations we have planned. If such is the case, as has been assumed by some writers, then we should expect the sedimentation rate to decrease during treatment with aspirin, salicylamide or sodium salicylate, and to increase with PAS. By the same token these changes in sedimentation rate would be expected to occur because of the mere presence of these substances in the blood, and not because of any real change in the underlying pathological process.

However, the results of the present study indicate that the relationship between the effects which can be produced *in vitro* and those which are observed *in vivo* is not so simple as has been thought to be the case in the past. The points in favour of this conclusion are as follows: (i) ordinary therapeutic levels of sodium salicylate do not affect the sedimentation rate of fresh blood; (ii) the effect of salicylate becomes apparent only when blood has aged, the effect being greater when salicylate is added after, rather than before, aging; and (iii) the reduction in sedimentation produced by aging and by salicylate is diminished, and in some experiments completely prevented, if blood is kept in motion while it ages—a procedure which imitates the circulation of blood in the body. In the light of these findings it cannot be assumed that the mere presence of salicylate in the blood-stream during the treatment of patients will reduce the sedimentation rate. It may be that some property of blood responsible for abnormally rapid sedimentation alters during storage in the test tube—an alteration which is accelerated by heat, by stagnation and by salicylate—and that the phenomenon is due entirely to the artificial circumstances of the experiments and is irrelevant to the effects produced by these drugs in the body.

Summary.

1. Sodium salicylate has practically no effect on the sedimentation rate of fresh blood. It has a pronounced inhibitory effect on that of blood which has been heated and kept stagnant. This effect is reduced if the blood is agitated during heating.

2. Sodium *p*-amino salicylate caused an increase in sedimentation rate under the same conditions.

3. Aspirin and salicylamide have effects similar to sodium salicylate.

4. It is suggested that the effects produced *in vitro* have no relation to what may be expected to occur *in vivo* during therapy.

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FOLIC ACID AND REPRODUCTIVE EFFICIENCY.

By D. F. LAWSON and C. N. DEGARIS,
Women's Hospital, Melbourne,
AND
J. H. BOLTON,
Royal Melbourne Hospital, Melbourne.

WHEN discussing macrocytic anæmia of pregnancy, two of us (Lawson and Bolton, 1951) stated that we regarded a lymphocyte count of less than 1500 per cubic millimetre as presumptive evidence of folic acid deficiency. When folic acid was administered in such instances there was a pronounced subjective and objective improvement in the patient's condition.

We have continued to look for patients whose clinical picture suggested a folic acid deficiency, and as we continued to find them, we became impressed with the frequency with which we found it in patients who had bad obstetrical histories. By those with bad obstetrical histories, we mean patients who had had miscarriages, unexplained stillbirths, unexplained premature births, or full-time infants weighing less than six pounds.

By using a lymphocyte count of less than 1500 per cubic millimetre as an index of folic acid deficiency, we have in all a series of 77 patients, of whom 21 were *primigravidæ* and 56 *multigravidæ*.

TABLE I.

Observation.	56 Patients with Folic Acid Deficiency.		56 Normal *Multigravidæ* for Comparison.
	Folic Acid Not Given.	Folic Acid Given.	
Total number of pregnancies	145	53	138
Miscarriages	67	—	9
Premature births	23	8[1]	7
Stillbirths	9	1	4[2]
Small mature children	5	1	4
Abnormal children	—	1	2
Full-time children	41	42	106
Neonatal deaths	4	5[1]	2[2]
Pregnancy still not terminated	—	3	6

[1] Four of which were a set of quadruplets.
[2] Including one abnormal child.

Among the 56 multigravid women there have been 145 "untreated" pregnancies and 53 pregnancies during which the patients received folic acid therapy for the whole of, or the major portion of, their pre-confinement period.

The outcome of all these pregnancies is as listed in Table I and for comparative purposes the results of the last 56 pregnancies in apparently normal *multigravidæ* in our practice are presented.

We consider that these figures suggest that there is some relationship between a low lymphocyte count and/or folic acid deficiency, and the inability to carry a pregnancy successfully to term.

Conclusions.

1. Multigravid patients with a bad obstetrical background have shown a marked improvement in their reproductive efficiency when a folic acid deficiency is detected and corrected.

2. In these patients, folic acid therapy is an important supplement to the measures which we regard as necessary in every pregnancy—that is (*a*) a diet rich in protein and vitamins, (*b*) the maintenance of a high hæmoglobin level, and (*c*) adequate weight control.

3. We know that folic acid therapy will not prove the answer for all patients with a bad obstetrical history; but we do think that evidence of its deficiency should be looked for in all such patients.

Acknowledgements.

Dr. Lucy M. Bryce has carried out the blood examinations in all of these cases, and we are grateful to her for making her skill available to us.

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THE INFLUENCE OF HYALURONIDASE ON ABSORPTION FROM THE COLON.

By PAUL HAGEN and K. W. ROBINSON,
Physiology Department, University of Queensland.

THE usefulness of hyaluronidase in increasing the rate of absorption of fluids, sodium chloride, glucose and various drugs injected hypodermically is well established. In view of its mode of action, it would scarcely be expected

TABLE I.

Substance.	Amount Introduced.[1]	With Hyaluronidase.		Without Hyaluronidase.	
		Amount Recovered.	Difference.	Amount Recovered.	Difference.
Glucose	150 milligrammes.	144 144 158	−6 −6 +8	156 144 144	+6 −6 −6
Propionic acid	870 micromoles.	253 141 180	−617 −729 −690	141 168 102	−729 −702 −768
Methionine	510 micromoles.	462 418 462	−48 −92 −48	473 455 355	−37 −55 −155
"Casydrol"	810 micromoles.	660 660 (Animal died)	−150 −150	682 639 682	−128 −171 −128
Sodium chloride	58 milligrammes.	43 50 40	−15 −8 −18	44 43 44	−14 −15 −14

[1] The amounts were injected in a volume of four millilitres with the exception of glucose, in which case 2·8 millilitres of solution were introduced.

to increase the speed of absorption of substances administered by rectum. However, as claims have been made by those supplying hyaluronidase commercially that it is of value in increasing the speed of absorption of a rectally administered drug, we have investigated the effect of hyaluronidase on the absorption of chlorides (as sodium chloride), glucose, propionic acid (as its sodium salt), methionine, and amino acid hydrolysate ("Casydrol") from the colon of the guinea-pig.

Methods.

All guinea-pigs weighed 450 ± 20 grammes. They were anæsthetized with "Sodium Pentobarbital" injected intraperitoneally. The wall of the colon was cut at its junction with caecum and with rectum, so that the same segment of alimentary canal was used in each animal. It was washed through with physiological saline (except in the experiment on chloride absorption, when it was washed with distilled water) until the washings were quite clear. It was then firmly ligated at both ends. The solution being investigated was injected into the colon through a fine hypodermic needle, and the animal was then stitched up and left. At the end of two hours the colon was removed, the contents were washed out thoroughly and made up to volume; a measured quantity of this was used for analysis, all estimations being made in duplicate.

Glucose was estimated by the Somogyi Nelson method; propionate by acidification, steam distillation and titration with barium hydroxide; methionine and amino acid hydrolysate by formol titration; and sodium chloride by titration with silver nitrate, tartrazine being used as indicator.

Six guinea-pigs were used for each experiment, three receiving a measured quantity of the solution under test and three the same quantity with hyaluronidase (10 Schering units of hyaluronidase to 50 millilitres of solution). The quantities introduced and recovered are shown in Table I.

Results.

From Table I it is clear that glucose was not absorbed from the colon either alone or in the presence of hyaluronidase, and that the quantities of propionate, methionine, amino acid hydrolysate and chloride absorbed during the two-hour period were uninfluenced by the presence of hyaluronidase.

Acknowledgements.

We wish to express our thanks to Messrs. Schering A.G., Berlin, who supplied the hyaluronidase used in these investigations. This work was done with the aid of a grant from the Rural Credits fund of the Commonwealth Bank.

Reviews.

One Little Boy. By Dorothy W. Baruch, with the medical collaboration of Hyman Miller, M.D.; 1953. London: Victor Gollancz, Limited. 8″ × 5⅛″, pp. 252. Price: 13s. 6d.

THE editor of "The Saturday Review of Literature" writes of this book: "The most searching inquiry into the mind of a child requiring psycho-therapy that I know of. The clinical case-history of seven-year-old Kenneth, graphically and skilfully told, will open to the reader the inner world, not only of this child, but of all children."

The basic principle of psychotherapy is that symptoms disappear when their origin in actual experiences is clearly uncovered for the patient. It is then that we may speak of "catharsis", or the relief that comes from cleansing through understanding. Such symptoms are said to be "functional", since there are no actual lesions to cause them. Relief came to the boy from showing him that his asthma and other symptoms were functional, that is, without physical basis. Explanation, however, was complicated for this patient by a more fundamental fact continuously operating in his conduct, namely, an unconscious fixation upon his mother, and by an estrangement between his mother and father.

The author's task was to bring to light the hitherto unrecognized undercurrents. Of this task she gives a complete and painstaking account. In this readers will realize, again, both the reality of functional disorders and the effect of family relationships upon the mental health of its members.

Varicose Veins. By R. Rowden Foote; Second Edition; 1952. London: Butterworth and Company (Publishers), Limited. Sydney: Butterworth and Company (Australia), Limited. 10″ × 7″, pp. 258, with 186 illustrations, a few in colour. Price: 46s. 6d.

IN the reprint of his book on varicose veins, Rowden Foote gives some surprising statistics of their incidence. He quotes the *Bulletin of Health* figures (1946) that nearly 10% of patients admitted to hospital in the Emergency Medical Service of Great Britain during the war were sufferers from varicose veins and their complications. The book covers extensively the history, anatomy, physiology and management of varicose veins. Monoethanolamine oleate ("Ethamolin") is considered the most useful general purpose sclerosant, but phenol 2% in glycerin 30% is preferred.

It is apparent that the author belongs to the school of thought whose operative planning is governed by the hypothesis that immediate post-operative ambulation is necessary to avoid thrombosis and embolism. This makes obligatory the use of local anæsthesia. Other surgeons favour early instead of immediate ambulation. They are not bound by the same restrictions and consequently enjoy a larger choice of operation. They will find little new in this book in the section on operative treatment except perhaps the author's special nutmeg grater needle for abrading the interior of the vein. Scant attention is paid

to operations which require general anæsthesia, and such widely practised operations as the stripping of veins and even ligature of the external saphenous vein are barely mentioned.

The best and most valuable chapters deal with varicose ulceration, thrombophlebitis, and supportive and compression methods. The author draws attention to the frequent association of tineal infection with varicose veins and states equivocally that "compression treatment correctly chosen and correctly applied, has always proved to be adequate" for the cure of varicose ulceration. Lumbar sympathectomy for these cases is condemned. He advocates ambulation in the treatment of phlebitis of the small superficial veins of the leg and that "all cases of thrombophlebitis of the internal or external saphenous veins must essentially be treated as surgical emergencies" by operation. This practice is not yet followed in Australia, but the advice is sound. This is an excellent book and one which all interested in the subject should read.

Cardioscopy. By William Evans, M.D., D.Sc., F.R.C.P.; 1952. London: Butterworth and Company (Publishers), Limited. 10" × 7", pp. 162, with 207 illustrations. Price: 56s. 6d.

IN the compilation of this book Dr. Evans acknowledges the great debt which he, in common with many of us, owes to Sir John Parkinson whose voice can almost be heard as one reads some of the legends.

The text of this book is firmly based on a remarkably clear collection of X-ray reproductions which reflect great credit on the technical skill of Mr. Dicks. The legend is concise, pertinent and quite adequate for a book of this type. It is easy to criticize the author for various omissions, but to deal with the subject fully would necessitate a large volume which would immediately detract from the practical value of the book. He briefly mentions orthodiagraphy, teleradiography, kymography, tomography and angiocardiography before concentrating on his screen to see firstly the normal heart and then the displaced heart before studying the abnormal.

His method of labelling landmarks by figures on a white disk is disturbing to some, but it has its advantages: (i) There is no doubt what the figure represents. (ii) It is surely better for the student to look at the X-ray picture and endeavour to discover for himself that a certain number is the pulmonary artery and then confirm it by reference to the legend, rather than to see the shadow labelled "P.A." to save him from the trouble of thinking.

The illustrations are so comprehensive and clear that it is difficult to individualize. With pericardial effusion the author shows the stencilled border particularly well. With surgery advancing, his section on calcification of valves is interesting, as also is the series of plates demonstrating the sequence of changes brought about by mitral stenosis. Abnormal œsophageal displacement by the left auricle in various conditions is demonstrated and discussed. His pictures of cardiac aneurysms are gems.

This book will prove instructive to the student, valuable to the radiologist and essential to the cardiologist.

Anatomy of the Autonomic Nervous System. By G. A. G. Mitchell, O.B.E., T.D., M.B., Ch.M., D.Sc., with a foreword by James Learmonth, K.C.V.O., C.B.E., Ch.M., LL.D., F.R.C.S. (Edinburgh); 1953. Edinburgh and London: E. and S. Livingstone, Limited. 10" × 7", pp. 372, with 131 illustrations, some in colour. Price: 55s.

As the author points out, there is no recent account of the anatomy of the autonomic nervous system. This defect he sets out to remedy in a systematic fashion and as completely as our present knowledge permits. His own work in the past probably qualifies him as well as anybody for this task. The book opens with a brief historical survey of the subject and then passes in natural order to definitions, development and histological structure. The systematic account begins—logically or otherwise according to taste—at the cerebral cortex and works down through the hypothalamus to the brain stem. After digressions to consider blood supply, and the details of parasympathetic and sympathetic components, the author pursues his systematic way through the neck, thorax, abdomen and pelvis. Where he discusses regions which have been the subject of his own well-known studies, Professor Mitchell betrays a happily certain touch. Where he is dependent upon the publications of others the work takes on more of an air of rather laborious compilation. In some parts, too, despite the avowedly practical outlook, there is involvement in unnecessary theoretical discussion. This is particularly the case with the frontal lobes of the brain. The section on blood supply appears to be more detailed and extensive than the nature of the work merits. On the other hand, the book is well illustrated, documented and produced. The price is not excessive for these days, and the volume will provide a valuable source of reference for those taking an interest in this increasingly important branch of knowledge.

Monographs in Medicine: Series I. Edited by William B. Bean, M.D., with associate editors Morton Hamburger, M.D., John A. Leutscher, junior, M.D., and Stewart Wolf, M.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 664, with about 50 illustrations. Price: £6 9s.

A NEW SERIES of medical monographs has been started and the first volume is edited by Professor William B. Bean, of the Department of Medicine, of the State University of Iowa, United States of America. It is well produced and attractive and deals with a variety of subjects. To review this book in the usual way is impossible; fifteen articles would have to be described and comments would have to be made on them. The titles of the "monographs" will be given, and from these and the names of their authors readers will be able to grasp the scope of the volume and the likelihood of its being of value to them. Stewart Wolf writes on "Talking with the Patient". This should not really be dignified with the title of monograph.

W. B. Bean deals with "Precordial Noise Heard at a Distance from the Chest"; he discusses the subject in the light of 126 references. W. M. Wallace's contribution is "The Physiology of Body Fluids", discussed in terms of the disturbances seen in clinical medicine. C. T. Dotter and I. Steinberg deal with practical applications of angiocardiography; they have some useful illustrations. "Portal Hypertension" by Mary A. Payne and C. G. Child is a discussion excellently illustrated and based on 154 references. Henry Aranow's contribution on "Pheochromocytoma" has 201 references. Fred Plum deals with "Respiratory Failure in Neuromuscular Disorders". Max Michael's "Cortisone and ACTH in Infectious Process" gives a warning that these substances are powerful and must be used with caution; their profound effects on a variety of experimental and clinical infectious processes are in the main deleterious. C. H. Rammelkamp and F. W. Denny in "Prevention of Rheumatic Fever" discuss the use of penicillin, aureomycin and the sulphonamides, and draw attention to the limitations of their usefulness. H. E. Hamilton in "Amebiasis" deals with the clinician's responsibility and has 64 references to the literature. L. H. Schmidt in "The Present Status of the Chemotherapy of Human Malaria" gives a short but clear-cut account of the subject. R. W. Schlesinger deals with "The Seasonal, Arthropod-Borne, Virus Encephalitides" and has some "remarks" on their relation to the general problem of virus encephalitis. B. S. Leavell and W. A. MacIlwaine discuss "Sickle Cell Anæmia" and deal with no less than 481 references to the literature. R. W. Vilter and J. F. Mueller write on "The Growth and Maturation of the Erythrocyte"; the subtitle of their article is: "A Consideration of Some Mechanisms Responsible for Anemia and Correlation of the Clinical and Biochemical Response to Therapy." D. A. Karnofsky deals with "Chemical Agents Used in the Treatment of Inoperable and Far-Advanced Neoplastic Disease".

Pain Sensations and Reactions. By James D. Hardy, Ph.D., Harold G. Wolff, M.D., and Helen Goodell, B.S., with a foreword by Edwin G. Boring, Ph.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9½" × 6", pp. 450, with 130 text figures. Price: 70s.

J. D. HARDY and his fellow authors describe their studies of "the phenomenology of pain". These studies are centred about the efforts of the authors to apply exact quantitative measurement to pain. To do this they have devised and used a "dolorimeter", an instrument which is inaptly named, for it does not measure pain. It delivers variable and measurable quantities of radiant heat, which is focused upon the skin of the subject, thus causing pain. As the intensity of the heat is increased, the subject appreciates increases in the pain. The just-noticeable-difference between one pain intensity and the next is called a "jnd", and two of these make one "dol", and these are taken as the units for the exact measurement of pain. The authors, experimenting on each other, find that between "threshold" and "ceiling" pain, there are 21 "jnds" or ten and a half "dols". For measuring length, a unit, for example, the metre, is not of much use, unless we are sure that one metre is of the same length

as another. For reasons that are not stated, the authors assume that one "dol" is equal to another, not only in the scale of pains of the same quality in the same individual with the use of the same stimulus upon the same tissue, but from subject to subject, from tissue to tissue, and between pains of different qualities, caused by differing stimuli. It is also assumed that a subject can, by introspection, compare one pain with another, so that a woman in labour, who experiences the skin pain caused by the "dolorimeter", can state the quantity of her labour pains in terms of "dols". In this way labour pains are compared, one pain with the next and one patient with the next, and the effect of analgesics on these and on other pains is studied. Past pains are even measured from memory and expressed in "dols".

The authors refer to the "scepticism" and the "doubts expressed on all sides" about the validity of such measurements. However, they persist with their painstaking experiments, and many facts, commonplace to the clinician, are presented anew, decked out in scales and graphs and other regalia borrowed from exact sciences.

Although the reading of the experimental details is tedious, the book would be of interest to the special student. It contains over five hundred references to the literature of neuro-physiology and related subjects.

Sympathetic Control of Human Blood Vessels. By H. Barcroft, M.A., M.D., M.R.C.P., and H. J. C. Swan, Ph.D., B.S., M.R.C.P.; 1953. London: Edward Arnold and Company. 9" × 6", pp. 172, with about 80 illustrations. Price: 18s.

"SYMPATHETIC CONTROL OF HUMAN BLOOD VESSELS", by Barcroft and Swan, is a record of the researches of Barcroft's school into the control of blood vessels in skeletal muscle and skin with relevant material on the pharmacology of this system. There are also chapters on phæochrome tumours of the adrenal and the vaso-cagal syndrome. This is a lucid account of the information derived from skilful experiments on the intact and sympathectomized human being. It is indispensable to anybody conducting research on these problems and provides information essential to the understanding of many clinical conditions.

The production is excellent and the bibliography good. This volume makes one look forward to further monographs from the Physiological Society.

Social Psychiatry: A Study of Therapeutic Communities. By Maxwell Jones, M.D., M.R.C.P. (Edinburgh), D.P.M., and A. Baker, M.B., B.S., D.P.M., Thomas Freeman, M.D., D.P.M., Julius Merry, M.B., B.S., D.P.M., B. A. Pomryn, M.B., B.S., D.P.M., Joseph Sandler, M.A., Ph.D., A.I.S., and Joy Tuxford, with a foreword by Aubrey Lewis; 1952. London: Tavistock Publications, Limited, in collaboration with Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 9" × 6", pp. 206, with two text figures. Price: 18s.

"SOCIAL PSYCHIATRY" is an account of Dr. Maxwell Jones's experiments with group therapy which began with his collaboration with Dr. Paul Wood in the Effort Syndrome Unit at Mill Hill during the second World War. Dr. Jones later took charge of an ex-prisoner of war community, and latterly of an Industrial Neurosis Unit, the administration of which forms the main topic of this book. A hundred beds for this unit were set aside in a large psychiatric clinic. Here each psychiatrist has a panel of from 20 to 30 cases, and the staff includes two disablement resettlement officers from the Ministry of Labour, psychologists, occupational therapists and social workers. About ten patients are admitted per week and the average stay is from two to four months. The main qualification for admission is that the patient is a chronic unemployed neurotic who is willing to submit to treatment. All methods of psychiatric treatment are available—physical and psychotherapeutic except psychoanalysis—but more particularly Dr. Jones attempts to apply sociology and anthropology to existing psychiatric practice. The daily programme begins with discussion groups in which personal and community problems are ventilated by the patients and nursing staff under the chairmanship of one of the medical staff. To quote Dr. Jones, "we have paid special attention to communications throughout the entire Unit population, together with free discussion of any problem affecting the community". The urgent needs of manpower prompted these expensive experiments in rehabilitation. What of the results? Unfortunately the follow-up covered a period of six months only, but at the end of that time the psychiatric social worker reported that 41% of the patients comprising this most unpromising material were considered employable on the open market, while a further 25% were employable under sheltered conditions. Actually 53% of the discharged patients had been employed for at least twenty-five weeks out of twenty-six. In an addendum the handling of a disciplinary problem is reported in some detail and doubtless many readers of this book will base their judgements on Dr. Jones's methods on this last chapter.

The Practical Management of Pain in Labour. By W. D. Wylie, M.A., M.B. (Cantab.), M.R.C.P. (London), D.A.; 1953. London: Lloyd-Luke (Medical Books), Limited. 9" × 6", pp. 160, with 42 illustrations. Price: 18s. 6d.

THIS little book gives a comprehensive account of various methods which may be used for the production of analgesia and anæsthesia in obstetrics with safety to mother and child. Maternal and fœtal anoxia and their interrelationship are simply explained in the opening chapter. Then follows an account of the use and effects, with a careful assessment of the value and danger, of such drugs as pethidine, morphine, trichlorethylene, chloroform, ether, nitrous oxide *et cetera*. Where special apparatus is required for the administration of inhalational agents, it is described and illustrated. An excellent chapter on the relief of pain in normal labour expresses the author's preference for pethidine in the first stage and trichlorethylene or nitrous oxide and oxygen in the second stage. Abnormal labour and surgical obstetrics require special consideration, and therefore anæsthetic substances are discussed from the point of view of suitability in specific conditions, as also are the various techniques for the production of local anæsthesia and sympathetic block. Obstetricians and anæsthetists alike will appreciate this informative publication.

The Treatment of Diabetes Mellitus. By Elliott P. Joslin, A.M., M.D., Sc.D., Howard F. Root, M.D., Priscilla White, M.D., Sc.D., and Alexander Marble, A.M., M.D.; Ninth Edition; 1952. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 772, with 26 illustrations, one in colour. Price: £6 9s.

THE ninth edition of "The Treatment of Diabetes Mellitus" by Joslin *et alii* has appeared six years after the previous one.

The book is now smaller, but the arrangement of the chapters is practically the same. Each chapter has been thoroughly revised and present conceptions of the physiology of *diabetes mellitus* have been well expounded. Although treatment of the disease by dieting and insulin is dealt with from every aspect, emphasis is laid particularly upon a complete control of the diabetic state in order to abolish or delay the onset of vascular degeneration with the accompanying triopathy, neuritis, retinitis and nephritis.

The aim of all the teaching in the book is to ensure that a diabetic remains healthy, not only for ten years, but for twenty and thirty years after the diagnosis has been established and treatment commenced.

The chapter on the treatment of diabetes contains a concise account of the uses of the three kinds of insulin, the quick acting, the slow acting and the intermediate acting, and the last mentioned includes the latest neutral protamin Hagedorn insulin (N.P.H.) which is of advantage particularly for children. Correct emphasis has been placed upon altering the diet by way of giving more of the allotted carbohydrates later in the day when the more slowly acting insulin is used.

The chapters dealing with diabetic children and their later lives and with pregnancy complicating diabetes written by Dr. Priscilla White are full of most useful information gleaned from a vast experience. Dr. White produces statistical evidence to support her belief that hormone therapy, namely, intramuscular use of stilbœstrol and progesterone commenced during the first trimester, materially reduces fœtal mortality and maternal toxæmia in the diabetic mothers who have an abnormal endocrine balance. This contention has not gained universal acceptance.

There is an appendix containing a new and simple method of calculating diabetic diets. Some of the foods mentioned are not obtainable in this country, but this does not detract from its usefulness.

The vast amount of statistical detail dealing with every aspect of diabetes is of immense value to the research student and the specialist in diabetes, as also are the chapters dealing with the complications of diabetes and other diseases complicating diabetes.

This book is most comprehensive, and is suitable for the specialist or for the practitioner who is particularly interested in the disease, but, for the busy practitioner, it could not be recommended as a "handy guide" to the treatment of diabetes.

Notes on Books, Current Journals and New Appliances.

FOUR NEW MEDICAL JOURNALS.

DURING the last few weeks, four new American medical journals have been received from the British agents, Baillière, Tindall and Cox, Limited, of 7 and 8 Henrietta Street, Covent Garden, London, W.C.2.

The first number of the *Journal of the American Geriatrics Society* bears the date January, 1953. It is the official organ of the American Geriatrics Society. It will be issued every month, twelve issues constituting a volume. The issue comprises 76 pages, and contains eight original articles, together with a short history of the American Geriatrics Society and a review of current geriatrical literature. A few pages are devoted to the Society proceedings and to the International Association of Gerontology. The articles deal with many aspects of geriatrics. The subscription to the journal is 82s. sterling *per annum*.

The first issue of *The Journal of Histochemistry and Cytochemistry* bears the date January, 1953. It is the official journal of the Histochemical Society, and is published by The Williams and Wilkins Company. The journal will be published six times a year, and each volume will contain about 500 pages. It will be devoted to original and review articles relating to the development and application of histochemical methods. The first number contains five original contributions, and also a list of "histochemical titles" from current literature. The annual subscription is 60s. sterling *per annum*.

The first issue of *Applied Microbiology* bears the date January, 1953. It is published under the sponsorship of the Society of American Bacteriologists. It will be published every two months, and is designed for the publication of studies "orientated toward the application of microbiological sciences to the fields of industry, foods, sanitation, agriculture, and other areas involving the use or control of microorganisms, with the exception of the microbiological aspects of animal and plant disease". The first issue contains twelve original articles which occupy 59 pages. The annual subscription is 63s. sterling.

The fourth journal received is entitled *The American Surgeon*. The number received is marked Volume 19, No. 1, and bears the date of January, 1953. This journal is new in name only; it has for some years been published as *The Southern Surgeon*. It is the official publication of the Southeastern Surgical Congress of America and is now published as a general surgical journal. It will appear every month, and twelve numbers will comprise a volume of about 1200 pages. The object of the journal is stated to be the advancement of surgery. The first issue contains twelve original articles, together with an editorial on hypertrophic scars and keloids. The editor is Dr. Thomas G. Orr, of the University of Kansas Medical Center. The subscription is 82s. sterling *per annum*.

"FAMILY DOCTOR."

"FAMILY DOCTOR" for May, 1953, is attractive. There is "Family Reading", followed by a section "Mainly for Mothers"; then we have "Medicine and Health" with some odds and ends that are grouped under "Home and Family". Each section has its own particular punch. In "Little Thief" John Brandon shows how thieving which is rare for a child with a secure home relationship can be prevented. "Mrs. Jay" gives a striking lesson in how to overcome a post-operative deformity—we like "Mrs. Jay". We also confess to a liking for the little boy who illustrates the article "Satan Finds Mischief"—it is important to see that there are no idle hands and no idle brains, and then Satan is the only one to feel frustrated. The philosopher in this issue is restrained—in his advice about sweets and golf. Ursula Shelley shows that "Feeding can be Fun". "Family Doctor" continues to live up to its reputation.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Cardboard Giants", by Paul Hackett; 1953. London: Victor Gollancz, Limited. 8″ × 5¼″, pp. 316. Price: 15s.

An account by a layman of his experiences in a mental hospital, intended for the general public.

"Baillière's Nurses' Medical Dictionary", revised by Margaret E. Hitch, S.R.N., with a foreword by Cecil Wakeley, Bt., K.B.E., C.B.; Thirteenth Edition; 1952. London: Baillière, Tindall and Cox. 5½″ × 4″, pp. 514, with 474 illustrations. Price: 5s.

This useful little dictionary was first published in 1912.

"Pediatrics in General Practice", by James G. Hughes, B.A., M.D.; First Edition; 1952. New York: McGraw-Hill Book Company, Incorporated. 10″ × 7¼″, pp. 748, with 178 illustrations. Price: $14.00.

The author has endeavoured to condense into one volume "some of the practical aspects" of pædiatrics.

"The 1952 Year Book of the Eye, Ear, Nose and Throat (October, 1951-September, 1952)"; The Eye, edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.); The Ear, Nose and Throat, edited by John R. Lindsay, M.D.; 1953. Chicago: The Year Book Publishers, Incorporated. 8″ × 5¼″, pp. 456, with 120 illustrations. Price: $6.00.

One of the Practical Medicine Series of Year Books.

"Clinical Approach to Fevers", by C. J. McSweeney, M.D., F.R.C.P.I., D.P.H.; 1953. London: J. and A. Churchill, Limited. 7½″ × 5″, pp. 152. Price: 10s. 6d.

The book represents a course of lectures given to postgraduate students, mainly from Irish universities.

"The 1952 Year Book of Urology (November, 1951-October, 1952)", edited by William Wallace Scott, M.D., Ph.D.; 1953. Chicago: The Year Book Publishers, Incorporated. 8″ × 5¼″, pp. 372, with 79 illustrations. Price: $5.75.

One of the Practical Medicine Series of Year Books.

"The Surgical Clinics of North America"; 1953. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Chicago Number. 9″ × 6″, pp. 310, with 35 illustrations. Price: £6 *per annum* with paper binding, and £7 5s. *per annum* with cloth binding.

This volume consists entirely of a symposium on obstetrics and gynæcology; there are 22 articles with a foreword. The titles of the articles will appeal to the general practitioner.

"The Medical Clinics of North America"; 1953. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Chicago Number. 9″ × 6″, pp. 294, with 32 illustrations. Price: £6 *per annum* with paper binding, and £7 5s. *per annum* with cloth binding.

The volume consists chiefly of a symposium on gastrointestinal diseases; there are eighteen articles, dealing with a variety of important subjects. There is one additional article on clinical and experimental hypoglycæmia.

"The National Health Service: A Guide for Practitioners", edited by Max Sorsby, L.M.S.S.A., with a foreword by Allen Daley, M.D., F.R.C.P.; 1953. Edinburgh and London: E. and S. Livingstone, Limited. 8½″ × 5½″, pp. 280. Price: 12s. 6d.

The aim is to give "a factual account of the scope and the day-to-day working" of the National Health Service in Great Britain.

"Plastic Surgery at The New York Hospital One Hundred Years Ago: With Biographical Notes on Gurdon Buck", by Herbert Conway, M.D., and Richard B. Stark, M.D., with forewords by John Hay Whitney, Stanhope Bayne-Jones, M.D., and Frank Glenn, M.D.; 1953. New York: Paul B. Hoeber, Incorporated. 8½″ × 6″, pp. 120, with 29 illustrations. Price: $5.00.

Dr. Buck "was among the first to recognise that restoration to maximum participation in the community is only possible when disfigurement has been reduced to the greatest possible extent".

The Medical Journal of Australia

SATURDAY, JUNE 13, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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THE PROBLEM OF ADJUSTMENT IN THE ARCTIC.

ON more than one occasion over the years discussions have taken place in these columns on the adjustment of the white man to life in the tropics. From the time when a spirited discussion took place on that subject at the Australasian Medical Congress at Brisbane in 1920, the question has arisen again and again. Australians have shown that the white man can live and work in the tropics and can enjoy good health. At the other end of the scale we have the Arctic and the Antarctic continents. Until recently, the interest of most people in this part of the world has centred round the work of expeditions sent north or south for scientific purposes. Australians may yet have to prove that they can live and work in the Antarctic regions. People nowadays talk in what appears to be a serious fashion about journeys to the moon. It is much less fantastic to suppose that man will some day explore the Arctic and Antarctic regions for industrial purposes. Australians may some day have to show the world that they can live and work in Adélieland. In the Northern Hemisphere, the Eskimos live and move (not over too wide an area) and have their being. They have become quite accustomed to their environment, and their main preoccupation, if one can believe what is written about them, is the acquisition of food. By all accounts they are finding this more and more difficult for reasons which need not concern us in this place. During recent years aeroplanes have been used for military purposes in the Arctic regions, and it is quite likely that the advent of war would make Arctic flights almost an everyday experience. All these considerations invest with particular interest an article recently published by T. J. Boag, of Montreal, Canada, entitled "The White Man in the Arctic".[1]

Boag read his paper at the annual meeting of the American Psychiatric Association in May, 1952. Of special interest is the discussion of the paper by Sir Hubert Wilkins, the famous Arctic explorer. Boag has carried out a series of observations which cover two types of situations. The first set of observations dealt with small, isolated settlements, and the second with groups of men camping out in tents and igloos in the winter. An attempt was made to assess the stresses impinging on these people and the ways in which the individual and the community reacted to them. Dealing first with environmental stresses, Boag points out that some of them are common to life on isolated stations in other parts of the world. The climatic stresses consist of cold, wind, fog and snow, separately and in combination. Any wind at all makes cold a great deal more trying than it would otherwise be, and in the barren lands high winds are common. It is interesting that in the Canadian barren lands deep snow is rarely a problem, as the total precipitation is small and snow soon becomes firmly packed by the wind. Boag states that, below the treeline, there is a more friendly and secure "feel" than above this level. Sir Hubert Wilkins pointed out that when walking over the hard-packed snow in the Arctic, a man feels the same sense of relief as he might feel in the city when he walks on a road after having crossed a ploughed field. This is one of the points on which soldiers could be instructed if they had to be subjected to such new and strange conditions. Temperatures are not significantly lower further south than in many parts of Canada, but winter lasts longer, there being snow on the ground from approximately October to June. To these factors have to be added the long hours of darkness through the winter. The isolation is similar to that experienced in many parts of the world. At the same time, the isolation is not as real as it was in the years before the Second World War ,as many of the settlements are now visited periodically by aircraft; some of them are visited regularly, and almost all of them can be reached by aircraft in case of emergency. Boag, discussing settlements, states that it is apparent that most of the inhabitants who have permanent quarters are not directly subjected to climatic factors as a main factor, but retire from them into the security of their accommodation. Sir Hubert Wilkins stated that this was the core of the problem. He added that the direct effect of cold and climate generally on military personnel and equipment during operations was very considerable. Boag points out that an occasional man will spend a whole winter in his quarters without leaving them to visit neighbours half an hour's walk away, and that quite commonly, men will visit their neighbours only on a couple of occasions during the winter. He points out that, with the onset of winter, men are progressively thrown into close contact, and that strong interpersonal relationships develop which may be concerned in later difficulties. Curiously, tensions reach a peak, not in the depths of winter, but at the end of winter when weather is usually good. Many of the differences of opinion seem to arise over ill-defined responsibilities, rights and privileges. Many of the people concerned know that this kind of trouble may arise and are afraid of any actual hostility; the result is suppression, repression, and withdrawal from situations in which trouble threatens to break out openly.

[1] *Am. J. Psychiat.*, December, 1952.

Boag also discusses this subject in regard to groups of men camping out in the winter. The accommodation of these men consists of tents and of snow huts or igloos. When the members of a party are broken up and allotted to several igloos, another set of barriers arises. Boag states that maintenance of comfort in an igloo depends on keeping it closed once the occupants are inside. He states that it is a surprisingly sound-proof structure, and that once it is sealed, the residents are effectively cut off from what is going on outside. In these circumstances, contacts between different sections of the party tend to be limited to the hours when everyone is out in the open. Sir Hubert Wilkins claims that the door of a properly built snow house is never closed or sealed. The top of the door is below the level of the living quarters, and so long as the venting of the house is properly controlled, the excessive cold cannot enter. He thinks that to seal the door introduces several unsatisfactory elements; the average white man in such surroundings seems imprisoned. Only when it is sealed is an igloo sound-proof. One can understand that if the door is left open, a feeling of restriction may be avoided, and any noisy activity going on outside may be heard. However, whether the door of an igloo is sealed or not, the loneliness of its occupants must be great. Boag refers to the restriction imposed on sight and hearing by the heavy clothing and hoods which have to be worn. This clothing constitutes a psychological as well as a physical barrier. In some expeditions which have gone from Australia, some of these facts have been noted; but we have it on good authority that when the major section of one expedition was thrown together for a long time in a restricted space, no irritability or quarrels were manifested. In this instance, all the members of the expedition were young men and all were university graduates—they had the same kind of physique and much the same mental outlook and training. Probably the predominating factor would be the personality of the leader and the way in which he arranged his schedule of the group's activities.

Boag had something to say about a programme for further researches. He thinks that more field investigation of group and individual situations in detail is required, "for further delineation of significant factors in the stresses imposed and the reactions to them". If surveys are to be made, they will entail a good deal of travelling, and Boag thinks that the method could be used only as a follow-up to previous detailed studies. He also declares that another aspect of the problem has to do with the criteria for selection of men for service of this kind. Sir Hubert Wilkins states that to the mentally balanced man, if he is protected, the stormy period should afford feelings of satisfaction and gratification, a feeling of satisfaction at being able to avoid the fury of the storm, somewhat akin to what might be felt when listening to the rain pattering on a roof. In his opinion, the problems of the white man in the Arctic are not all psychiatric or psychological, but rather problems of indoctrination, training and administration. In general, he states that the proper man for service in the Arctic is the average, common-sense man with keen sensitivity and normal physical and mental ability. "The Arctic is no place for the subnormal, a difficult place for the supernormal, and impossible for the supersensitive man who lacks control."

Current Comment.

AN UNSOLVED PROBLEM OF BIOLOGY.

SINCE we are the products and exemplars of a universe which is cyclical so far as we know it, we are prone to assume that age or the inevitable changes associated with time are equally linked with deterioration and decay. The melancholy Jaques merely voiced common philosophy when he said:

> And so from hour to hour we ripe and ripe,
> And then from hour to hour we rot and rot,
> And thereby hangs a tale.

From a consideration of this act of aging two important problems arise. One is of the highest sociological importance, and concerns the progressive increase of older people in the community due to longer survival. The other is of special interest to doctors, physiologists, pathologists, geneticists and planners of the so-called health schemes. It raises the question whether senescence consists of a decline in vitality, not necessarily concerned with aging, or may be regarded as the fulfilment of our cyclical existence so neatly expressed by Jaques, in which, we should observe, there is wrapped up the concept of maturation extending well into adult life, and that of deterioration creeping upon us even at an early age.

P. W. Medawar, Jodrell Professor of Zoology and Comparative Anatomy in the University of London, delivered an inaugural lecture at University College on December 6, 1941, dealing with the problem of the origin and evolution of aging.[1] This lecture, recently published, puts forth a thesis in a closely knit web of proven science, argument and speculation. The author early points out the need for some definition of the terms "aging" and "senescence", or at least some explicit understanding of what is meant by them. "Aging" in this lecture means simply the process of growing old or, with that curious gentleness of the comparative degree in English grammar, "growing older". "Senescence" is taken to mean a measurable decline of vitality. This may be estimated by observation of the anatomical and physiological phenomena at different ages, such as wound healing, acuity of hearing, muscular coordination *et cetera*. It may be more clearly measured by statistical methods, such as the compilation of a life table, which should be called a death table, as Medawar correctly observes. He points out, too, the fallacies of such tables. The vulnerability of humans to the hazards of life differs greatly during life, and the age of twelve years, after the drop following the dangerous neo-natal period, may be regarded as the actuarial prime of life, even though the development of stamina and of muscle performance, like the ossification of certain bones, is delayed till twice that age. Professor Medawar, the biologist, and the Shakespearian philosopher agree in emphasizing this curious paradox.

A little later in the lecture the author shows that senescence is of twofold origin, innate senescence, and the senescence caused by the sum of the effects of recurring injury, stress or infection. Only in experimental animals can this difference be always clearly established. The author invokes the parallel of a set of one thousand test tubes exposed to stresses of daily use in a laboratory, and points out that sheer age increases exposure to hazard. There is, however, another most important consideration, that the older age-groups have a smaller overall reproductive force, not because of lowered fertility, but because of their poverty of numbers. Natural selection in this test tube scheme will suffer lowering of its force with increasing age. This brings us to the genetic aspects of the problem, and Medawar lays some stress on what he calls "overt action" of unfavourable heredity factors, a postponement of which may be enforced by the action of natural selection. To doctors this part of the argument is made more interesting by the use of Huntington's chorea as an example. It is rightly pointed out that there is a

[1] "An Unsolved Problem of Biology", H. K. Lewis and Company, Limited, London.

possible fallacy in any assumption concerning the age of onset, and consequent delay in action of the genes, for investigation has shown that this disease may emerge at various ages, some very youthful indeed. This genetic aspect was dealt with admirably by M. F. Hickey in his Jackson Lecture delivered in Brisbane on September 7, 1951, and recently published in this journal on May 9, 1953. He points out that Huntington's chorea is "a remarkable example of variation in the expressivity of a gene shown in the variable age of onset".

Medawar finally sums up three agencies which may play a part in the evolution of what he calls "innate" senescence. These are (*a*) the inability of natural selection to counter the weakened effect of mutation when the genes express their effect in the older age groups, (*b*) the fact that a harmful gene may be eliminated by postponement of its time of action, and (*c*) that natural selection may help in such postponement. The author does not claim to present a fully documented theory, but he makes some suggestions which evoke our interest in the genetic side of this subject of senescence. Earlier he disposes of some of Weismann's conjectures in senescence, but in a final summing up is inclined to relent with regard to the broad outlines of his ideas. Since this is all that we as average medical readers are able to discuss with any conviction, we may pay tribute to the advantages of awareness of this problem, even though, as Medawar's title states, it is still not completely solved.

ATYPICAL CONGENITAL HÆMOLYTIC ANÆMIA.

THE causes of hæmolytic anæmia fall naturally into four groups in relation to the life of the erythrocyte: abnormalities of the erythrocyte itself, either congenital or acquired; destructive factors introduced into the erythrocyte, for example, the malaria parasite; hæmolytic substances present in the plasma, including immune bodies, bacteria and chemicals; excessive phagocytic activity on the part of the reticulo-endothelial system. In practice it is not always easy to attribute a particular case of hæmolytic anæmia to a single cause, or to place it in one of these groups, but it seems reasonably clear that all the known hereditary forms of hæmolytic anæmia belong to the first group, that is to say, they are due to inherited abnormalities of the erythrocytes. Three varieties of congenital hæmolytic anæmia are well established as clinical entities. Though each has a variety of names, they will be recognized under the names used in Whitby and Britton's well-known book "Disorders of the Blood": congenital hæmolytic icterus (acholuric jaundice), sickle cell anæmia (African anæmia) and Mediterranean anæmia (target-oval cell anæmia, or Cooley's anæmia). Apart from these, a number of more or less distinct types have been described in recent years. A notable recent contribution to the subject is a report by J. V. Dacie, P. L. Mollison, Nancy Richardson, J. G. Selwyn and L. Shapiro of twelve cases of congenital hæmolytic anæmia, each differing in a significant respect from the three typical forms. It is stated that, for various reasons, the families of the patients in the series have not been exhaustively studied and in only six of the ten families is there evidence of a familial incidence. However, Dacie and his colleagues have grounds for believing that the disorder in each case is genetically determined. The cases have been classified under five headings. The first group, consisting of five cases, is described as "non-spherocytic congenital hæmolytic anæmia". It is notable that four of the five patients in this group underwent splenectomy but obtained no apparent benefit. The second group of four cases is given the general description of "variants of hereditary spherocytosis". Three patients in this group were members of the same family. One of the three most severely affected underwent splenectomy with considerable benefit. The other three "groups" consist of only one patient in each: a patient with macrocytosis and leg ulcers, a patient with a variant of familial elliptocytosis, and a patient with "triangular" red cells. In the first of these last three cases, the blood picture, with macrocytosis and extreme variation in red cell size and shape, differentiated the condition from what was otherwise a fairly typical example of congenital hæmolytic icterus. Splenectomy appeared to produce no real benefit. The patient with elliptocytosis had a clear family history, but, as is usual with this condition, his anæmia appeared to provide the exception rather than the rule. He obtained great benefit from splenectomy. The last patient had a very suggestive family history. Splenectomy failed to affect the clinical course of her condition.

Dacie and his colleagues describe the morphology of the red cells of their patients as "remarkable for its diversity". Almost all the recognized deviations from the normal were found in the blood groups, as well as deviations not commonly met with. The striking variety in size, shape and staining of erythrocytes is well brought out in photomicrographs that accompany the article. As might be expected, splenectomy, even if it did not appreciably affect the clinical course of the disease, invariably caused some modification in the blood picture. All the disorders in the series were thought to be due to inherited defects of the erythrocytes, which resulted in a diminished life span *in vivo*. *In vitro*, in some cases at least, the cellular abnormality was reflected in an accelerated rate of spontaneous hæmolysis and an abnormal alteration in osmotic fragility on incubation of the blood at 37° C. A summary of relevant literature is included in this paper, and the atypical cases of congenital hæmolytic anæmia reported by various authors are classified into four groups: non-spherocytic congenital hæmolytic anæmia, atypical hypochromic congenital hæmolytic anæmia, elliptocytic congenital hæmolytic anæmia and atypical hereditary spherocytosis.

The study of these cases of hæmolytic anæmia should help towards the clarification of a class of anæmia that is still by no means fully understood. Indeed, the description of these cases in terms of their "atypical" features in relation to the generally recognized clinical entities serves particularly to emphasize that our present classification is arbitrary and a concession to relative ignorance.

[1] *Quart. J. Med.*, January, 1953.

RESULTS OF SURGICAL TREATMENT FOR PULMONARY STENOSIS OR ATRESIA.

BETWEEN November, 1944, and October, 1950, Blalock and his associates at Baltimore operated on 1000 patients for the alleviation of pulmonary stenosis or atresia by means of systemic-pulmonary anastomosis. Helen B. Tausig and S. R. Bauersfeld[1] have followed these patients to March 1, 1952; in all cases the follow-up period was at least eighteen months, and in some cases it was seven years. The findings make encouraging reading. Results of operation were classified as good (773 patients), fair (39 patients), unimproved (31 patients) and died (157 patients). A good result means that cyanosis was much less and oxygen saturation of arterial blood rose to between 75% and 90%, usually with corresponding hæmatological and subjective improvement. A fair result means that improvement occurred, but cyanosis and polycythæmia of varying degrees persisted. In the follow-up analysis the patients were divided into those with tetralogy of Fallot and those with atypical malformations. The first observation of particular interest is that those with an initially good result did far better on a long-term basis than those with an initially fair result; the latter, in turn, did better (though only slightly) than those who were unimproved by operation. The results in the tetralogy of Fallot group indicate that most patients with this condition are able to adjust themselves to the altered circulation. A second operation was sometimes of value, but the mortality rate was high. The results in the atypical formation group were rather less favourable. In the whole series the incidence of subacute bacterial endocarditis has been low; apparently the additional anomaly does not seriously increase the patient's susceptibility to this complication.

[1] *Ann. Int. Med.*, January, 1953.

Abstracts from Medical Literature.

PATHOLOGY.

Pseudomembranous Colitis following Aureomycin and Chloramphenicol Therapy.

L. REINER, M. J. SCHLESINGER AND G. M. MILLER (*Arch. Path.*, July, 1952) state that pseudomembranous colitis may follow aureomycin or chloramphenicol medication. The mucosa of the colon is the seat of two characteristic changes usually, but not necessarily, occurring together: surface exudation ("simple" pseudomembrane) and stromal (diphtheritic) necrosis. The "simple" pseudomembranes may be muco-epithelial, fibrino-muco-epithelial, fibrino-purulent or muco-fibrino-purulent. The stromal (diphtheritic) necrosis is an essential constituent of "compound" pseudomembranes. It varies in lateral extension and in depth. Healing occurs by sequestration of the pseudomembranes and simultaneous epithelial regeneration. The deeper the stromal (diphtheritic) necrosis, the more distorted, atrophic or scarred is the resulting healed mucosa. The colonic mucosa is subject to repeated injury by the original agent. Pseudomembranous colitis follows aureomycin therapy, not only after oral but also after intravenous administration. The incidence of the pseudomembranous colitis shows a female sex preponderance. Neither intestinal pathogens nor fungi are demonstrated. A variety of vitamins neither prevents nor cures the pseudomembranous colitis. The clinical manifestations are not characteristic. Diarrhœa is common but not constant. The mechanism by which aureomycin and chloramphenicol produce pseudomembranous colitis is obscure.

Malignant Synovioma.

C. J. E. WRIGHT (*J. Path. & Bact.*, July, 1952) states that malignant synovioma has proved to be much more common than was at one time thought. In all, 24 cases were so diagnosed in the department of pathology of the University of Leeds during the years 1936 to 1949 inclusive, but a review of the material over the forty-year period 1910 to 1949 has disclosed 23 additional cases. In every case the situation of the tumour was compatible with a synovial origin; the lower limb, especially the region of the knee, was the commonest site. All 47 patients have been followed up; only six of them are still alive and have survived for any considerable length of time. Two have survived twenty-four years and thirteen years respectively after local excision of the tumour, but in the other four, who have all survived for more than nine years, the tumours have recurred. Thirty-five patients developed generalized metastasis, and in this group the average survival period after the primary operation was two years. Synovial differentiation is the most characteristic histological feature. Pseudoepithelial and sarcomatous structure are often intimately blended, and the only useful classification is according to the degree of differentiation. The tumours are then divisible fairly readily into two groups, the rare well-differentiated and the more common poorly differentiated forms. The former may be of non-giant-cell type, the synovium-like spaces being lined by cells closely resembling glandular epithelium, or of giant-cell type. The five patients in this group were among the six long-term survivors in the series. The prognosis associated with the poorly differentiated tumours, whether of ordinary type (39 cases) or of giant-cell type (three cases), was bad; only one patient is still alive and has survived for any considerable length of time, and even in this case the tumour has recurred twice. Of 24 patients suffering from malignant synovioma, for whom local excision was the only initial treatment, 17 developed local recurrence. In nine cases lymph-nodal metastasis was present without other sign of spread. Prognosis is related to histological structure more than to mode of surgical treatment. All the patients with well-differentiated tumours are still alive and have survived for as long as twenty-four years since the primary operation. Local excision or biopsy is recommended as the first procedure, depending on the size, situation and degree of encapsulation of the tumour. If it is of the poorly differentiated type immediate amputation should follow histological diagnosis, but if it is uniformly well differentiated, wide local excision should be adequate. All transitions may be observed between the benign giant-cell synovioma and the various forms of malignant synovial tumour. The existence as an entity of malignant giant-cell synovioma admits of no doubt.

The Ocular Lens and Radiation Injury.

SOLBERT PERMUTT AND FRANK B. JOHNSON (*Arch. Path.*, January, 1953) have demonstrated polysaccharide-protein complexes in the capsule, cement substance, epithelial cells and fibres of the normal ocular lens. They state that after irradiation of the lens, the following changes take place: (i) swelling of the capsule, the lenticular fibres and the cement substance; (ii) increased reaction with the Hotchkiss stain in these areas; (iii) increased solubility of the polysaccharide-protein complexes in these areas. These changes are interpreted as resulting from depolymerization of the polysaccharide-protein complexes of the lens. The relation between the depolymerization and the pathogenesis of radiation cataracts is discussed.

Hyaline Material with Staining Reaction of Fibrinoid in Renal Lesions in Diabetes Mellitus.

LEOPOLD G. KOSS (*Arch. Path.*, December, 1952) describes a lesion consisting of an accumulation of a hyaline material with the staining reactions of fibrinoid in kidneys of diabetics with various degrees of renal sclerosis. He states that this material accumulates in the glomerular tufts, glomerular capsules and basement membranes of proximal convoluted tubules and is in every respect comparable to the material found in the walls of some arterioles showing hyaline arteriolar sclerosis and with tubular casts of inspissated protein. It is believed that the material represents precipitated protein accumulating in spaces created by splitting of the basement membranes of the kidney. The lesion appears to be in every way comparable to arteriolar hyaline sclerosis. The hyaline-fibrinoid lesion is not strictly specific for diabetes. Its apparent specificity is only quantitative, since the amount of the hyaline-fibrinoid material accumulating in severely damaged diabetic kidneys is greater and its distribution wider than in other renal diseases. The hyaline-fibrinoid lesions appear to be an important component in the renal lesions associated with *diabetes mellitus*. It is, according to the author, quite distinct from the nodular lesion described by Kimmelstiel and Wilson. It is possible that occurrence of the hyaline-fibrinoid lesion may contribute to renal failure.

Metastatic Cystadenocarcinoma of the Ovary.

JAMES B. HUTCHESON (*Arch. Path.*, September, 1952) reports the case of a woman, aged sixty-two years, who had papillary cystadenocarcinomata of both ovaries removed thirty-three years prior to death. She remained asymptomatic until six months before death when œdema of the right lower extremity, swelling of the abdomen and constipation developed. *Post mortem* a large, dense, honeycombed tumour was found occupying the right pelvic and iliac regions and extending into the iliac bone and caecum. The liver and iliac, aortic and mesenteric lymph nodes contained metastatic tumour. Histologically, the tumour represented papillary cystadenocarcinoma similar to but more anaplastic than the growths originally removed. The author states that the case is remarkable for the longest recorded period of survival of a patient with this type of ovarian carcinoma in whom the growth was present at the time of death; it provides an extreme example of the ability of cancer cells to lie dormant for long periods and then to grow again.

Polysaccharide Nature of Corpora Amylacea.

HOWARD D. STEELE, GORDON KINLEY, CECILIE LEUCHTENBERGER AND ETHEL LIEB (*Arch. Path.*, July, 1952) state that *corpora amylacea* of the prostate, the lung and the brain contain a polysaccharide with a 1,2 glycol grouping. The concentration of the polysaccharide varies in individual corpora in the prostate and the lung. Nucleic acid, either as desoxyribosenucleic or ribosenucleic acid, is not present. It is suggested that the *corpora amylacea* of the prostate and the lung are formed from inspissated secretion and that there is no evidence of cells as a constituent.

Unsuspected Carcinoma with Thyroid Disease.

JOHN M. BEAL, GEORGE L. SCHOLNICK AND G. ARNOLD STEVENS (*Arch. Surg.*, December, 1952) present data on the incidence of carcinoma in 200 consecutive patients with thyroid disease subjected to operation. The material was obtained in a non-endemic goitre area. In the series 15 cases of carcinoma (7%) were encountered, and all 15 occurred in non-toxic nodular goitre. Of 133 patients with clinically non-toxic nodular goitre, five (3·75%) were found to have unsuspected carcinoma. Of the 85 patients who presented a clinical impression of

solitary nodular goitre, four were later found to have carcinoma, while only one of the 48 who were thought clinically to have multinodular goitre was subsequently found to have carcinoma. The age distribution of the patients with carcinoma did not differ significantly from that of those with non-toxic nodular goitre without carcinoma. The incidence of carcinoma was approximately four times greater in men than in women in this series.

MORPHOLOGY.

Sebaceous Glands on the Hand.

S. G. JOHNSEN AND J. E. KIRK (*Anat. Rec.*, April, 1952) state that, in view of the comparative scarcity of data regarding the sebaceous glands in the dorsal region of the hand and the minimal fatty secretion observed in this area, they carried out a thorough histological study of this region. They found that each hair is supplied with one small uniobular sebaceous gland. The number of glands per square centimetre of skin varies from nil to 50, the glands being found in greater number in the region adjacent to the wrist. Measurements of 135 sebaceous glands showed that 80% of the glands have a volume less than 0·00050 cubic millimetre and that the mean size of the glands is 0·00044 cubic millimetre.

Cell Rests in the Region of the Fourth Ventricle.

R. J. BRZUSTOWICZ AND J. W. KERNOHAN (*Arch. Neurol. & Psychiat.*, May, 1952) examined 253 cerebella, not involved by tumours, for the presence of cell rests. The tænia of the fourth ventricle (ponticulus) and cerebellar nodulus were the most frequent sites of cell rests. The cerebellar white matter was involved less frequently, while the anterior, posterior and inferior medullary vela were involved least frequently. Cell rests occurred most frequently in stillborn term infants, premature infants and fœtuses. The incidences of rests in the cerebella of adults, children and infants were approximately the same. The frequency of occurrence of rests in males was greater than in females in term infants less than one week old, in stillborn term infants, in premature infants and in fœtuses. In the older age groups, there was little difference in the frequency of rests in males and in females. Because the roof structures have been thought to be the site of numerous cell rests, many authors have ascribed the origin of tumours involving the fourth ventricle to these rests. The results of this study indicate that the ponticulus of the medulla, forming the most inferior border of the fourth ventricle, contains as many cell rests as, or more than, the nodulus, which forms part of the posterior vermis of the cerebellum. With regard to classification, mixed-cell rests were encountered most frequently. No correlation was noted between the frequency of cell rests of a certain histological type occurring in a specific structure and the frequency of tumours of the same histological type arising in that structure. The region of the nodulus in the posterior vermis was the most frequent site of origin of the gliomata in this study. This region was also the most frequent site of mixed-cell rests.

Histology of Egyptian Mummies.

W. GRAF (*Acta anat.*, 1949, 8: 236) found that in Egyptian mummies the connective tissue showed the highest degree of histological preservation, the collagenous and elastic fibres being easily recognizable structures. The cartilage had a highly atypical appearance, with entirely destroyed chondrocytes and preserved but distorted bundles of collagenous fibrils. The bony tissue showed persistence of the collagenous matrix. In muscles and nerves the sarcolemmata and perineural sheaths respectively were the only details that could be identified. Red blood corpuscles were the sole cellular elements found in the sections examined. In the histological sections the connective tissue fibres as well as the red blood corpuscles showed typical staining reactions. Examination was also made of ancient Swedish skeletons, but the author found no parallelism between their historical age and the degree of histological preservation. Those skeletons found in dry earth (gravel) generally showed the best preservation. Among the structures observed was articular cartilage, in which there were remnants of chondrocyte endoplasma and a weak but quite typical metachromatic reaction of the chondrine. In the spongy bone, recognizable bone marrow cells were seen; and in the Haversian canals of the compact bone, there were blood vessels and red blood corpuscles. Different pictures of the disintegration of the bone were met with, signifying that the resistence against destruction is not homogenous. Thus the inner and outer basic lamellæ and in addition the lamellæ bordering the Haversian canals were as a rule least destroyed. Among the early signs of destruction observed was a widening of the lacunæ and canaliculi, which when sufficiently enlarged fused to greater defects. In other specimens it was found that the collagenous fibre system of the bone was distorted and broken up into fragments, which might be entirely separated or might show more or less complete continuity. The process of disintegration could not be traced in its whole by means of studying the different stages. It has also been demonstrated that cells enclosed in the Haversian canals are well preserved when studied a relatively short time after burial. In a bone unearthed after one year, the intravascular leucocytes could be easily identified and eosinophile granules of usual brilliance were observed.

Cardiac Conduction System.

W. M. COPENHAVER AND R. C. TRUEX (*Anat. Rec.*, December, 1952) studied histologically hearts of sheep, monkey and man, in an attempt to clarify some of the controversial points regarding the atrial portion of the cardiac conduction system. Sino-atrial nodal fibres were identifiable in each case, but no atrial Purkinje fibres were found in sheep hearts, where identification of these fibres is easily made, and in only one of 23 human specimens were atrial elements observed which simulated the Purkinje fibres which are found in the conducting system in the ventricles. The authors doubt the existence of a specialized conduction pathway between the sino-atrial and atrio-ventricular nodes, and support the prevalent view that the atrio-ventricular node is composed of slender fibres similar to those of the sino-atrial node, and that the atrio-ventricular nodal fibres are continuous on the one hand with ordinary atrial muscle and on the other with the ventricular muscle by way of the atrio-ventricular bundle of His. The authors assert that their findings are contrary to those of other recent authors who state that there is no morphological evidence to support the myogenic theory of conduction.

Effects of X Rays on Development.

J. G. WILSON *et alii* (*Am. J. Anat.*, January, 1953) exposed rat embryos on the ninth day of gestation to doses of 25*r* to 400*r* of X rays directly through an abdominal incision in the mother. Lead plates were arranged so that selected embryos could be irradiated while the mother and remaining embryos were shielded. The irradiated and non-irradiated embryos were removed one to eight days later or at term and compared as to weight, the presence of malformations and the rate of intrauterine mortality. Exposure to 25*r* resulted in microphthalmia in a very few instances, but otherwise had no effect on embryonic development. Dosage with 50*r* caused a slight retardation in the rate of growth and resulted in ocular maldevelopment or anophthalmia in 34% of animals. The brain and spinal cord also were malformed in a few instances. Irradiation with 100*r* was followed by a considerable increase in mortality, and in the survivors caused considerable retardation of growth, which, however, became less pronounced as the post-irradiation interval increased. Some type of ocular abnormality was present in 90% of the animals. Malformations in other organs were less frequent, but still occurred with considerable regularity in the brain, spinal cord, face, heart and aortic arches. *Situs inversus* and anomalies of the urinary system were encountered occasionally. Many of these defects were found as often in term animals as in embryos removed at earlier ages; but anomalies involving the central nervous system and the cardio-vascular system were less frequent in term animals and appeared to be associated with, if not responsible for, the high rate of prenatal mortality. Virtually all embryos receiving 200*r* died within four days after treatment. and a large majority of those given 400*r* died within twenty-four hours. The over-all effects of irradiation on the ninth day of gestation were more severe than those following treatment on the tenth day, owing presumably to the greater susceptibility of the less-differentiated ninth-day embryo to mitotic delay and genic alterations.

Aplasia of the Male Prepuce.

T. JAMES (*J. Anat.*, October, 1951) describes four cases of failure of development of the male prepuce with hereditary background definitely established in two cases. The author was unable to find any reference to this condition in the literature; but he believes that the condition cannot be an uncommon one, and refers to a dispute in ancient times mentioned in the Talmud concerning a child who was born circumcised.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LXVI.
DISSEMINATED SCLEROSIS.

DISSEMINATED SCLEROSIS is one of the commonest organic diseases of the nervous system in Western Europe; and it is now becoming more common or is at least more often recognized in the United States, Australia and New Zealand. It develops, often as an acute disorder, especially in early adult life in individuals who are otherwise healthy. It follows an intermittent course characterized by the occurrence of active phases which appear at intervals of a few weeks or months to many years and last from a few days to several months. After each active phase there is usually clinical improvement with either no residual disability, some residual disability for the first time or an addition to the disability already present as a result of earlier acute phases. Under unfavourable conditions, the development of the disease may extend over many years and eventually lead to complete disablement in the majority of cases; but, under favourable conditions, the disease may be less severe, become apparently arrested at any stage and lead to total disablement in only 5% to 10% of cases.

Underlying this characteristic clinical course is the occurrence in successive crops over many years of multiple inflammatory lesions scattered irregularly throughout the brain and spinal cord. The lesions are disseminated in time as well as in space; and, as a result, the clinical study of the patient who may suffer from the disease must pay full attention to the details of the medical history of the patient over many years and to the symptoms and physical signs that indicate the occurrence at any time during that period of lesions in more than one part of the brain and spinal cord. The lesions may appear in any part of the brain and spinal cord from the cerebral cortex to the conus, but are most common around the ventricles of the brain, at the junction of the grey and white matter under the cerebral cortex and around the basal ganglia, around blood vessels, and in the optic chiasma and optic nerves. These common sites of involvement are related to the commonest clinical features of the disease. The disease itself is an acute one in its active phases and is regarded as a chronic one only because of the recurrence of active phases, the residual effects of the acute lesions and the summation of residual disabilities following the active phases.

The characteristic history and clinical features are illustrated by the following case, in which there was good resistance to the disease under the best conditions.

The patient, a man, aged thirty-three years when he was first seen, gave the following history. At the age of twenty-eight years he had for a few weeks the feeling that his right foot was swollen and that his right shoe was too tight. At the age of thirty-one years he had for three months the feeling that both lower limbs were frozen below the knees. Nine months before he came under observation he saw double with the images side by side on looking to the left, and recovered in a few days. A short time after that he developed coldness with numbness and tingling in the lower limbs up to the knees, and a tight feeling across the front of the abdomen. He continued to have these symptoms and also had frequency of micturition during the last six months. On examination of the patient the findings were as follows: a slight increase in resistance in the plantar flexors of both feet; the knee and ankle jerks active and abrupt; an absence of both plantar reflexes, but an extensor response on reinforcement; failure to feel vibration at the left ankle and faint appreciation of it only at the right ankle; occasional errors in recognizing changes of position of the toes of the right and left feet with the eyes closed; all other findings in relation to the nervous system normal. There were no objective variations in tactile, thermal or pain sensation. The patient improved rapidly and had no symptoms or physical signs during the following six years. At the age of thirty-nine years he developed soreness of the left eyeball on movement of the eyeballs and to the touch and became unusually tired, and some days later developed blurring of vision of the left eye. On examination of the patient, the left eyeball was slightly tender to the touch, there was some impairment of vision in the central portion of the left visual field, and all other findings in relation to the central nervous system were normal. These variations subsided within four weeks from their onset, and at the age of forty years the patient was free of symptoms and physical signs.

Within the framework provided by this case it should be possible to view in their right proportions the symptoms and physical signs of a disease which presents a moving and not a still picture. The data obtained from the clinical study of a large number of patients and summarized below must always be viewed in terms of time as well as of space.

The Presenting Symptoms.

The patient who appears with his first acute phase of disseminated sclerosis presents himself with symptoms which vary much in nature and position according to the site in which the first lesions are developing. Some of the common presenting symptoms are the following.

1. Spontaneous sensations such as tingling, pins and needles, numbness, coldness and less often burning appear constantly for a period of from a few days to a few weeks in the limbs, trunk or face. The phases of spontaneous sensations may be single and self-contained or multiple, affecting one part and then another and even a third part, with individual phases either separated by a short interval or overlapping one another in time. Common sites of spontaneous sensations are the following: one or other lower limb from the foot up to various levels as far as the hip; both lower limbs and the lower part of the trunk, often with a feeling of tightness around the trunk at the upper limit of the abnormal sensation or an even higher level; one or other upper limb from the hand up to various levels as far as the shoulder; one upper limb and then the other upper limb in the same manner; one upper limb and the face on the opposite side; the limbs and trunk on one side with or without the face on the same or opposite side.

2. A limb, a part of a limb or more than one limb may become slightly weak, sometimes within an hour or two. The weakness may involve an arm, a leg, an arm and a leg on the same or on opposite sides, both legs or both arms.

3. A limb or more than one limb may become limp and clumsy without obvious weakness, or unsteady, and shake during active movement.

4. There may be short phases of double vision usually lasting only a few days at a time and apt to be recurrent.

5. The vision of one eye may become blurred or partially lost with or without soreness of the eyeball on movement of the eyeballs or to the touch.

6. There may be a change in the emotional balance. The patient often becomes elated and may appear naïve and may manifest a greater freedom of emotional expression in the form of smiling and laughing; but occasionally it is to sadness with a tendency to weeping.

7. Vertigo or a disturbance of balance may appear, sometimes suddenly, occasionally with other symptoms such as vomiting, pain in the eye and limpness of the arm on the same side.

8. There may be impairment of control of the sphincters, usually of the bladder but occasionally of the bowel.

Less common presenting symptoms are as follows: double vision and ptosis with impairment of upward and outward deviation of an eyeball; facial paresis, which may recur on the same or on the opposite side; epileptic fits, which may develop slowly enough to appear to be focal in nature; pain in part of the territory of the trigeminal nerve; vomiting and other features of vagal disorder, often with vertigo; rapidly developing hemiparesis, sometimes with vasomotor changes in the limbs affected; slowly developing weakness of both lower limbs or of the arm and leg on one side.

Various combinations of these symptoms may appear, either together or in rapid succession.

The Clinical Features.

In active phases and even for many years during the course of the illness, the symptoms and physical signs may be readily recognized as those related to two or more foci of involvement of the nervous tissue, the site of which can be determined in most cases. When there have been several acute phases, however, and a summation of residual disabilities after them, it may not be so easy to identify all the sites of involvement responsible for the symptoms and physical signs.

1. Any of the following may be found in the cranial nerve territory. Diplopia is fleeting but often recurrent and not as a rule associated with a visible squint. Paresis of individual eye movements, either alone or in combination, is not frequent, though partial or complete paralysis of movements innervated through the third, fourth or sixth nerves and even complete external ophthalmoplegia may be seen. Paralysis of accommodation rarely occurs. Very occasionally pupils are seen which are sluggish or fixed on exposure to

light. Nystagmus is usually horizontal and lateral but rarely rotatory, depends upon alternations of straying of the eyeball from a position of lateral deviation and of volitional resumption of that position, and is common when cerebellar communications are involved. It may be vertical when the posterior longitudinal bundle is affected. Blurring of vision of one eye with pain in the eyeball in the early stages is common, having occurred in 25% of my own cases, and is due to retrobulbar neuritis. It is usually unilateral, but it may involve the other eye in another acute phase after a short or long interval. A central scotoma, complete or partial and sometimes limited to a defect in the appreciation of colour, is usually associated with it. Other variations in the visual fields, often temporary, due to involvement of central visual paths, may be found. The optic disc is usually normal with retrobulbar neuritis; but it may be swollen when the active process is just behind the optic disc. Slight and sometimes moderate swelling of the optic disc with early blurring or loss of vision is due to retrobulbar neuritis and not to increased intracranial pressure. At a later stage the optic disc may be pale on the temporal side, even when no clear history of retrobulbar neuritis has been obtained. Paræsthesiæ of the face and pain in the distribution of the trigeminal nerve, most often that of the first division, have already been mentioned. Facial paresis is not uncommon and may recur on the same or on the opposite side. Vertigo is common and may be associated with pain in the distribution of the ophthalmic division of the trigeminal nerve, vomiting and weakness with hypotonia of the upper limb on the same side as a result of involvement of the brainstem at the root of the restiform body. Articulation may be slow, spaced or slurred—sometimes as a result of incoordination of the movements concerned with speech when cerebellar communications are involved; sometimes as a result of spasticity when the upper motor paths are involved on both sides at a high level; and often as a result of both together and the attempt to overcome the ataxia.

2. In the motor field, paralysis of movement, with or without spasticity, both of the type and distribution characteristic of involvement of the upper motor path, may appear as slight to moderate monoplegia, paraplegia, hemiplegia, tetraplegia or weakness of both upper limbs or of an upper limb on one side and of a lower limb on the other side. In early cases and in those with the minimal residual effects of active phases, it is necessary to make observations in detail on the motor power and muscle tone to recognize the effects of involvement of the upper motor path. Amyotrophy is rare but does occur. Ataxia in the limbs is usually due to involvement of cerebellar communications, but it may depend partly or wholly, especially in the lower limbs, upon loss of appreciation of changes of position due to lesions in the spinal cord. With ataxia of cerebellar origin, tremor appears on active movement, and there is an alternation of straying of the limb from and of replacing it on the direct path when approaching the part to an object. With ataxia due to loss of appreciation of changes of position, the defect becomes apparent only when visual impressions are not available.

3. Spontaneous sensations of the type already mentioned may appear in various parts either in a fugitive form or for a few weeks. Pain is less common, but it does appear and varies from the aching that arises from persistent attempts to use a weak limb to characteristic spinal nerve root pains due to involvement of sensory paths within the spinal cord. Objective sensory changes are less obvious; and there is often in an acute phase a distinct contrast between the severity and constancy of the spontaneous sensory phenomena and the absence or slight degree only of impairment of tactile, thermal and pain sensation. With involvement of the spinal cord, however, there may be inability to feel vibration at the ankles or to recognize changes of position of the toes and even of the lower limbs with the eyes closed. In the presence of active lesions in the spinal cord, especially in the cervical enlargement, flexion or extension of the neck, either active or passive, may produce tingling or a feeling like electricity in hands or feet or both. This is an effect observed with only one other condition, traumatic changes in the spinal cord from head injury with forcible flexion or extension of the neck.

4. Variations in the reflexes, to which much attention has been paid, depend upon the presence, degree, position and age of lesions involving the upper motor paths; and a difference between the Mayer thumb reflexes on the two sides, inequality or absence of the abdominal reflexes, activity with or without abruptness of the tendon reflexes and the presence of extensor plantar reflexes are significant only in relation to the anatomical involvement and not in relation to the pathological changes producing it. They do become clinically important, however, when an attempt is being made to determine whether fleeting and transient symptoms, which are so common in the early stages of disseminated sclerosis, are accompanied by unequivocal evidence of anatomical involvement. Reflexes concerned with the bladder and bowel may be released from higher control by lesions in the spinal cord or brainstem. Two features commonly found in the patient with disseminated sclerosis should be noted: (*a*) It is often possible in the presence of lesions in the spinal cord to elicit an extensor plantar reflex with a slight, moderate or even pronounced flexor withdrawal movement of the lower limb in response to a slightly painful stimulus to the skin at various levels up to that of the lesion itself. (*b*) In patients who come under attention late in the course of the disease and have been exposed to the effects of arsenic over a long period, the more distal tendon reflexes, such as the ankle jerks, may be lost.

5. Emotional rather than mental changes are common. The patient may be cheerful and even euphoric; and often smiles or laughs unnecessarily on replying to an inquiry, responding to a sensory stimulus or carrying out a movement at command. With this variation there may be an appearance of naïvety. Pathological laughing is rare. Occasionally the patient is depressed rather than elated, and may weep easily. Often there are rapid fluctuations in mood. There may be a sense of mental and physical well-being, which is in decided contrast with the presence of the symptoms and physical variations, and even with the general fatigability which is now known to be a common feature of an acute phase of the disease. The grouping of increased emotional display, euphoria and a sense of physical well-being may be the first clinical expression of the disease; it is presumed to be due to the presence of periventricular lesions which would not reveal themselves in any other way; and is a much more common feature of the disease at any stage than the Charcot triad of nystagmus, scanning speech and intention tremor, which is found in less than one-quarter of the advanced cases in which there has been a steady summation of residual disabilities following successive acute phases.

6. There may be disturbances due to involvement of long sympathetic paths in the brainstem and cervical portion of the spinal cord, and of communications from higher controlling levels of the sympathetic nervous system in the hypothalamus or cerebral cortex; these are features of paralysis of the cervical sympathetic, vasomotor changes, glycosuria and vomiting. It should be understood, however, that small differences in the size of the pupils and palpebral apertures on the two sides are very common, especially in patients with emotional tension, and need not be due to damage to nervous structure.

Observations on the clinical pathology are needed in order to make a diagnosis in only rare cases; they are mentioned here only to prevent confusion with other neurological conditions in cases in which they may have been made. In one-quarter to one-half of the cases, usually during an acute phase of the disease, there is an increase in the number of cells per cubic millimetre in the cerebro-spinal fluid, in nine-tenths of them to less than 10, and in the others to a higher number and sometimes as many as 500. The cells are usually small round cells with some polymorphonuclear cells and an occasional endothelial cell, or entirely small round cells. The Lange colloidal gold curve is of the "paretic" type in about one-quarter of the cases; and may show minor variations ranging from the "luetic" type up to the "paretic" type in another one-quarter of the cases or more.

The Diagnosis.

The diagnosis of disseminated sclerosis must always be made in a positive manner on the basis of the data obtained and not by a process of exclusion; not only because it is the only proper clinical approach to a problem involving the central nervous system, but also because the latter method involves, as well as unnecessary biochemical and radiological examinations, procedures such as lumbar puncture, cisternal puncture, and even pneumoencephalography and myelography, which may be harmful to the patient with the disease, aggravate an acute phase of the disease and lessen the chance of the patient's recovering from it with little or no disability. The clinical history must include not only that of the recent phase of illness but also that of the life of the patient, with attention to previous illnesses in terms of symptoms as well as of the names given to them by the patient, so that any earlier phase of the disease may be recognized. The patient must be examined in minute detail in relation to the nervous system as well as generally; the findings, both symptoms and physical signs, must be grouped in terms of physiological functions; and the data so grouped must be interpreted in terms of the site or sites of anatomical involvement. If, by studying the patient in this way, it is found that there has been more than one phase of neurological involvement even over a long period of years, and the symptoms and physical signs indicate the occurrence of lesions in more than one part of the central nervous system,

the diagnosis should present itself. Even without a history of more than one active phase, there should be no great difficulty, for the appearance of clinical features due to lesions in the common sites of involvement and the presence of other characteristic features of the disease should make it relatively easy to recognize the nature of the pathology. Retrobulbar neuritis in an otherwise healthy individual is almost invariably due to the disease; early blurring of vision of one eye with slight swelling of the optic disc is usually due to retrobulbar neuritis far forward; euphoria and an increase of emotional display with evidence of a recent lesion in the brain, brainstem or spinal cord suggest that there may have been earlier periventricular lesions of the disease; and a distinct contrast between the severity of persistent spontaneous sensations and the paucity of objective sensory variations in the same territory is a characteristic feature. Though the earlier neurologists said that the first diagnosis usually made in a case of disseminated sclerosis was one of hysteria, the possibility of such an error should be slight if the clinician collects all the data, groups them in physiological terms, interprets them so far as he can in anatomical terms, understands that the error arises most often through trying to diagnose both disseminated sclerosis and hysteria by a process of exclusion, and recognizes that even transient early symptoms can be related to their physiological basis and anatomical background, both disseminated sclerosis and conversion hysteria have their own characteristic positive features, and clinical features due to anatomical involvement may be found together with characteristic phenomena of conversion hysteria.

The risk of confusion with focal conditions of the brain and spinal cord, such as tumour, should be small after detailed clinical study and removed entirely by securing the help of the best clinical methods available. *Neuromyelitis optica* is characterized by the occurrence a few weeks apart of acute myelitis and retrobulbar neuritis with swelling of the optic discs, though either condition may appear first or alone; it is a more severe condition than disseminated sclerosis in any early acute phase; and it attacks both eyes and produces greater damage to the optic nerves and to vision. Disseminated encephalomyelitis occasionally leads to difficulty, but it is non-recurrent. Epidemic encephalitis and *myasthenia gravis* are confused with disseminated sclerosis only in the absence of adequate clinical study.

The Course and Prognosis.

The course and prognosis of disseminated sclerosis in an individual case cannot be foretold during a first or an early acute phase, but can often be estimated when there have been several acute phases and there is the opportunity of determining the resistance of the patient to the disease and the presence and degree of any residual disability after each phase. In most cases the clinical course proceeds by way of recurrent acute phases over many years with short or very long intervals between them. In such cases there is, under the worst conditions, the prospect of ultimate disablement in the majority of cases; and, under the best conditions, that prospect in only 5% to 10% of cases. Under good conditions patients are living full and useful lives with practically no disability twenty and twenty-five years after the onset even when several acute phases have occurred. In a few cases, the occurrence of the individual acute phases is not so obvious, the condition seems to progress steadily, and the ultimate prognosis is usually bad. In most cases, however, in which the resistance is good and the conditions are favourable, it should be recognized that the condition of the patient may become stabilized at any stage. In every case the factors which appear to lessen the resistance of the patient to the disease and to provide the opportunity for the appearance or aggravation of acute phases are as follows: overwork and constant fatigue; intercurrent illness; the administration of general anæsthetics with or without surgical intervention; artificial termination of pregnancy; pregnancy itself; procedures which vary even temporarily conditions within the skull and spinal canal, such as lumbar and cisternal puncture, pneumoencephalography, myelography and surgical procedures, which are usually exploratory, on the brain and spinal cord.

The Treatment and Management.

The treatment of the patient who suffers from disseminated sclerosis depends upon the recognition and application of basic principles which should not be ignored. Failure to recognize them often leads to the use of procedures that may be harmful, inadequate care of the patient during the acute phase, the unnecessary application of methods of treatment which may be harmful but are claimed by the uncritical and inexperienced to be useful, and the use of unnecessary and even harmful methods of treatment for residual disabilities. In few conditions in medicine and particularly in neurological medicine is it more necessary for the clinician to apply in the management of the patient the first principle of all medical care, *primum non nocere*.

1. During an acute phase the patient should be treated and managed as one who is suffering from an acute illness. Rest, and even rest in bed in severe phases, is imperative. At least all activities which may aggravate the fatigability of the patient and, in particular, all activities which put stress on any functions that are affected must be avoided. Care must be taken not to be misled by the euphoria, the detachment from the presence of disability and fatigability, and the sense of well-being of the patient. Lumbar puncture should be omitted, a limitation difficult to obtain in hospitals in which a routine of so-called "investigation" often without adequate clinical study has become established; and other procedures which are too often a substitute for full clinical study must not be undertaken. Tonic support of the patient may be begun. Activity should be resumed only gradually after it is certain that the acute phase has subsided.

2. In the absence of any known specific treatment for the disease and whatever agent or factors produce it, it is necessary to rely both during the acute phase and after it upon building up and maintaining the resistance of the patient. Arsenic in the form of *Liquor Arsenicalis*, four minims three times a day, may be given regularly during the first year and on alternate months during the second year after an acute phase. Justification for the use of arsenic over a long period is provided by the observation that, as a rule, any subsequent acute phases are, if they should occur, relatively slight. There is, however, no good reason for giving intensive courses of arsenic intravenously when there is no evidence that it has any direct effect on the factors causing the disease.

3. The management of the life of the patient suffering from disseminated sclerosis involves accepting responsibility for giving advice and guidance on social, economic and human problems as well as on medical and therapeutic matters. The patient should live a normal life and follow an ordinary occupation, but recognize his limitations in regard to the effects of overwork, fatigue and any residual disabilities he may have, and learn to live and work within them. In unfavourable cases and when residual disabilities are becoming severe, it is necessary for him to curb his ambitions, limit his activities in regard to gainful occupation, refrain from accepting further economic and social responsibilities, and maintain interest and occupation, either gainfully or not, within the limits of what he can do. The frequency with which young women come under attention, already married or about to be married, in an early acute phase of the disease calls for advice in regard to marriage and pregnancy. The occurrence of an early acute phase cannot be regarded as a barrier to marriage, especially when it leaves no residual disability and other acute phases do not follow closely upon it. Nor can it be regarded as a contraindication to pregnancy, though it is advisable that the risk of a possibly unfavourable effect of pregnancy should not be accepted more than once or twice. When there have been several acute phases in rapid succession and residual disabilities are established, marriage is undesirable and pregnancy should be forbidden. When a woman with some disability becomes pregnant there is no particular indication for terminating the pregnancy, for that procedure may do more to lessen the resistance of the patient than the pregnancy itself. When disabilities are already established, general anæsthetics and surgical procedures should be limited to those that are essential. Intercurrent illness demands extra care, especially during the convalescent period.

4. Complications such as epilepsy require the usual treatment for such conditions, and others such as neuralgia and spinal nerve root pains should, whenever possible, be treated by conservative methods.

5. The management and treatment of the patient with residual disabilities demand patience, judgement and protection of the patient from unnecessary and harmful forms of treatment and the attentions of those with unscientific and quack remedies. Various forms of "shock" treatment, artificially induced pyrexia, injections of liver preparations and the wasteful use of vitamin preparations are all unnecessary. The "shock" treatments and artificial pyrexia may be even harmful. Physiotherapy, properly controlled and limited to judiciously administered massage, passive manipulation, application of heat to stiff joints and training in active movements, may help to maintain the usefulness of limbs and prevent spasm and abnormal postures. The various forms of electrotherapy may be harmful and are definitely contraindicated when there is involvement of upper motor paths.

In short, the treatment of the patient at this stage should be limited to maintaining as much useful function as possible, providing whatever help he may need to lessen the risk of

fatigue, supporting his morale, and protecting him from the host of quack and pseudo-scientific remedies that are sure to be forced upon his attention by uncritical and often ignorant claimants. It demands a balanced sense of proportion and fine judgement in regard to the patient's therapeutic, economic and social needs.

I. M. ALLEN,
Wellington, New Zealand.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Western Australian Branch of the British Medical Association was held at the Cottesloe Civic Centre, Perth, on March 27, 1953, the President, Dr. John Day, in the chair.

The annual meeting was combined with a buffet tea which replaced the annual dinner. This change was made by the Council in the hope that the arrangement might be more acceptable to members.

PRESENTATION TO DR. F. W. CARTER.

The President explained that Dr. F. W. Carter was present as guest of honour. He spoke of the immense amount of work which Dr. Carter had done for the Branch, and enumerated the many positions which Dr. Carter had filled. He said that there were few who had given such long and onerous service to the Association. Members of the Branch thought that Dr. Carter should be presented with some token of their appreciation. On behalf of the members, he then presented Dr. Carter with an inscribed silver tray. In thanking the members for the gift, Dr. F. W. Carter said that he had always enjoyed British Medical Association work. He spoke of some of the negotiations in which the Federal Council had been concerned, and said that he was gratified that Dr. D. E. Copping had succeeded him as Branch representative. He thanked the President for his generous remarks, and said that he would treasure the Branch's gift as one of his proudest possessions.

MINUTES.

At the conclusion of the buffet tea, the minutes of the previous meeting were read and confirmed on the motion of Dr. Lindsay Gray, seconded by Dr. Robert Linton.

THE ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council, which was received and adopted, is as follows.

The President and members of the Council of the British Medical Association have much pleasure in presenting the fifty-fourth annual report of the Branch for the year ending March 27, 1953.

Membership.

The membership of the Branch has increased during the twelve months under review, and the total number of members is now 514, compared with last year's total of 498. Gains were: New members 29, transfers from other Branches 36, total 65. Losses were: Transfers from this Branch 35, resignations 6, membership lapsed 2, deaths 6, total 49. A gain of 16.

Obituary.

With deep regret we record the deaths of the following members which occurred during the year: Dr. K. G. Aberdeen, Dr. J. Vere Arkle, Dr. G. W. Barker, Dr. F. S. Butler, Dr. H. G. Caulfield and Dr. F. P. Guilfoyle. The sincere sympathy of the Branch is extended to the families of these late members.

Meetings.

In addition to the annual meeting, eight general meetings of the Branch were held, including a film evening and a clinical evening. A special meeting was held in February at which Dr. E. G. Saint addressed the profession on "Plans and Prospects of the Clinical Research Unit, Royal Perth Hospital". The subjects of the meetings were as follows: April: "Medical Aspects of Atomic Warfare". Film evening. May: "Modern Status of the Hospital", Sir Victor Hurley. June: "Some Problems in Allergy in General Practice", Dr. H. Breidahl. July: Clinical evening, Princess Margaret Hospital. August: "Treatment of Ischæmia of the Legs by Artery Grafting", Professor Rob. September: "Insulin and Diabetes", Professor C. H. Best. October: "Cervical Disk Lesions", Dr. D. Brinton. November: "Common Eye Diseases in General Practice", Dr. G. A. Lamb. February: "Plans and Prospects of the Clinical Research Unit, Royal Perth Hospital", Dr. E. G. Saint.

Council Meetings.

Twenty meetings of the Branch Council were held. Record of attendance is as follows:

Dr. J. L. Day (President)	18
Dr. A. L. Dawkins (Vice-President)	15
Dr. J. H. Stubbe (Honorary Treasurer)	14
Dr. G. N. Barsden (Honorary Secretary)	18
Dr. C. W. Anderson (Honorary Assistant Secretary)	18
Dr. F. W. Carter (Federal Representative until December 31, 1952)	16
Dr. H. L. Cook (Federal Representative as from January 1, 1953)	18
Dr. D. E. Copping (Federal Representative as from January 1, 1953; also Press Liaison Officer)	7
Dr. B. W. Buttsworth (Chairman of Convocation)	9
Dr. B. O. Bladen (Chairman, Contract Practice)	6
Dr. L. I. Henzell (Commissioner of Public Health)	10
Dr. P. W. Atkins (Councillor)	17
Dr. D. M. Clement (Councillor)	18
Dr. S. E. Craig (Councillor)	18
Dr. G. C. Moss (Councillor)	17
Dr. A. B. Wilson (Councillor)	19

Office-Bearers and Councillors.

The following members have been elected as office-bearers for 1953: *President*, Dr. A. L. Dawkins; *Vice-President*, Dr. J. H. Stubbe; *Honorary Treasurer*, Dr. D. D. Keall; *Honorary Secretary*, Dr. G. N. Barsden; *Honorary Assistant Secretary*, Dr. C. W. Anderson; *Chairman of Convocation*, Dr. B. W. Buttsworth.

The following members have been elected as the five ordinary members of Council for 1953: Dr. D. M. Clement, Dr. B. C. Cohen, Dr. S. E. Craig, Dr. J. A. Gollan, Dr. A. B. Wilson.

At this juncture we would like to express our warm appreciation of the work of the retiring Councillors.

Federal Council.

Dr. F. W. Carter and Dr. H. Leigh Cook have again represented the Western Australian Branch on the Federal Council during the past year, and on behalf of all members we would like to express to them our very sincere appreciation of their untiring efforts during a very strenuous year. We would like to take this opportunity of welcoming Dr. D. E. Copping, who with Dr. Leigh Cook has been elected Federal Representative for 1953.

Library.

The report of the Library Committee will be heard in full, but the Council wishes to sincerely thank the chairman of the Library Committee, Dr. H. S. Lucraft, the committee and the library staff for their cooperation and for the increasing facilities of the library, this being made possible by the keen interest and attention to this most important factor of our Association. We should like to thank members who made donations of books and periodicals, all of which are very much appreciated. We would also like to place on record our appreciation of the sum of £750 granted to the Association for the Library by the Medical Board.

Representation.

The Branch was represented by the following members at various meetings and conferences during 1952:

Council of the British Medical Association.—Dr. Miles Formby.

Federal Council of the British Medical Association in Australia.—Dr. F. W. Carter, Dr. H. L. Cook.

University Advisory Board in Medicine.—Dr. H. H. Stewart.

Australasian Medical Publishing Company, Limited.—Dr. F. W. Carter.

State Health Council.—Dr. B. O. Bladen, Dr. H. L. Cook, Dr. A. B. Webster, Dr. M. F. Williams.

Nurses' Registration Board.—Dr. Love, Dr. Le Souef.

Dental Board.—Dr. I. O. Thorburn.

College of Dental Science.—Dr. I. O. Thorburn.

Workers' Compensation Act.—Dr. N. H. Robinson.

Underwriters' Joint Committee.—Dr. H. M. Hill, Dr. G. B. Maitland.

Saint John Ambulance Association.—Dr. A. L. Dawkins.

Optometrists' Registration Board.—Dr. D. D. Paton.

Sex Education (Parents and Citizens' Association).—Dr. I. O. Thorburn.

Protection of Practices.—Dr. F. W. Carter.

National Health Service Disciplinary Committee.—Dr. F. W. Carter, Dr. H. L. Cook, Dr. H. J. Gray, Dr. H. S. Lucraft.

Dr. P. C. Thomas represented the Editor of THE MEDICAL JOURNAL OF AUSTRALIA.

Public Health.

We wish to take this opportunity of thanking Dr. Linley Henzell, Commissioner of Public Health in this State, for his cooperation and efforts on our behalf during the past year and to assure him of this Association's cooperation in the future.

Australasian Medical Publishing Company, Limited.

Dr. F. W. Carter has been the director nominated by this State for the past twelve months and, as may be expected, has carried out the necessary duties in his usual very efficient manner. The Branch Council is most appreciative of having such a zealous representative.

Post-Graduate Committee.

During 1952 the Post-Graduate Committee under the chairmanship of Dr. Bruce Hunt had a very successful year. The committee arranged the scientific papers for the monthly British Medical Association meetings and assisted members in their post-graduate study by sponsoring visiting lecturers from overseas, sponsoring members to England and providing ships' surgeon appointments.

An important feature again this year was the holding of a medical lecture course, and arrangements are now in train for a 1953 course.

The post-graduate week held in August was a great success. Three eminent overseas lecturers addressed the profession during the week. These were: Professor Charles Best, co-discoverer of insulin, who gave several very interesting lectures on this subject; Dr. Avery Jones, the British authority on diseases of the alimentary tract, who gave several lectures and two clinical demonstrations; Sir Gordon Gordon-Taylor, the eminent British surgeon, who gave informative talks during this week.

Dr. Denis Brinton, Professor Le Gros Clarke and Professor Rob, all eminent men from overseas, gave interesting and informative lectures during the year.

Publicity.

As a result of the work done by our Press liaison officer, Dr. D. E. Copping, better relations and closer liaison between the Council and the managing editors of all papers has been maintained to the mutual advantage of both sides. We take this opportunity of thanking Dr. Copping for his extremely capable efforts on our behalf.

Contract Practice.

Under the chairmanship of Dr. B. O. Bladen, the Contract Practice Committee has finalized a number of contract agreements in the mining and timber districts. There were two round table conferences with members of the Friendly Societies Health Service Council, which were to the mutual benefit of both sides.

Timber and Mining Agreement Fees.—Standard Timber Mill Agreement, 96s. *per annum*; Special Timber Mill Agreement, 132s. *per annum*; Extraordinary Timber Mill Agreement, 120s. *per annum*; Mining Agreement, 92s. *per annum*. Special rates other than above are applicable to certain agreements where particular circumstances pertain.

Social.

The annual medico-legal golf match was held at Lake Karrinyup Country Club in October, followed by dinner at the club. The medicos won again this year on the return of the best four ball card by Dr. J. P. Ainslie and Dr. S. E. Craig. The B.M.A.C. Trophy, played for by members of the Association, was won by Dr. Ainslie. Our congratulations are extended to these men.

Convocation.

Two meetings of convocation have been held during the period under review. The main items for discussion were National Health and Pensioner Medical Services, *Pharmaceutical Benefits Act* and hospital and medical benefits funds. These meetings were well attended by country and metropolitan group representatives.

Staff.

The Council wishes to express to the office and library staff its sincere thanks for their cooperation and help to the Association during the year.

FINANCIAL STATEMENT.

The balance sheet as at December 31, 1952, and the income and expenditure account for the twelve months ended December 31, 1952, were received and adopted on the motion of the Honorary Treasurer. The statements are published herewith.

BRITISH MEDICAL ASSOCIATION, WESTERN AUSTRALIAN BRANCH.

Statement of Assets and Liabilities as at December 31, 1952.

LIABILITIES.	£ s. d.	£ s. d.
Subscriptions Paid in Advance ..		21 0 0
Sundry Funds:		
Building Fund	175 5 0	
Car Badges Account	29 10 0	
Entertainment Fund	3 15 9	
Library Donations Account ..	6 14 2	
Cyril Bryan Library Fund	230 0 11	
Nelson Bequest	18 4 7	
Post-Graduate Committee Account	609 13 9	
Post-Graduate Grant Account ..	21 9 8	
Publicity Fund	147 12 0	
		1,242 5 10
Accumulated Reserve Account of General Funds:		
Balance at January 1, 1952 ..	2,336 2 6	
Less Excess of Expenditure over Income	61 9 10	
		2,274 12 8
		3,537 18 6
Bank of New South Wales—Overdraft		182 11 5
		£3,720 9 11

ASSETS.	£ s. d.	£ s. d.
Sundry Debtors:		
Members' Subscriptions in Arrears	89 5 2	
Medico-Legal Golf Account ..	6 10 0	
	95 15 2	
Funds Overdrawn—		
Medical Board (Library) Grant Account	33 16 5	
		129 11 7
Investments:		
Australasian Medical Publishing Company, Limited—		
Debentures: Series "A"-"D"	345 0 0	
Debentures: Series "E" ..	917 17 6	
	1,262 17 6	
Commonwealth Bonds	1,410 0 0	
British Medical Agency Company	9 0 0	
		2,681 17 6
Fixed Assets:		
Furniture and Fittings	956 17 9	
Less Depreciation at 5%	47 16 11	
		909 0 10
		£3,720 9 11

BRITISH MEDICAL ASSOCIATION, WESTERN AUSTRALIAN BRANCH.

Income and Expenditure Account for Twelve Months Ended December 31, 1952.

EXPENDITURE.

				£	s.	d.	£	s.	d.
To Subscriptions:									
(a) Members' Journals:									
(i) Aust. Med. Pub. Co., Ltd.	£493	0	0						
Less Building Fund Debentures—Series "E"	£123	5	0						
				369	15	0			
(ii) B.M.A., London				627	4	11			
							996	19	11
(b) Federal Council, Sydney							504	0	0
(c) Medical Benevolent Association							449	8	6
(d) Flying Doctor Service							1	1	0
(e) Historical Society of W.A.								12	6
							1,942	1	11
„ Costs of Administration—									
Advertising				4	8	6			
Auditors' Fees				31	10	0			
"B.M.A. House" Expenses				15	15	0			
Depreciation				47	16	11			
Duplicating				11	6	7			
General Expenses				24	13	10			
Legal Expenses				5	7	6			
Library Expenses				244	1	4			
Medico-Political Expenses				78	11	4			
Postages and Petty Cash				151	12	10			
Printing				17	14	5			
Rent				200	6	5			
Stationery				69	13	10			
Telephone				74	7	2			
Salaries:									
Secretary	£686	16	8						
Senior Typist	£428	0	0						
Junior Typist	£268	4	5						
Librarian	£526	15	0						
Library Assistant	£273	6	0						
	£2,183	2	1						
Less B.M.A.C. Refund	£131	0	6						
				2,052	1	7			
							3,029	7	3
							£4,971	9	2

INCOME.

	£	s.	d.	£	s.	d.
By Members' Subscriptions				4,814	17	6
„ Interest on:						
(a) Commonwealth Treasury Bonds	48	7	9			
(b) *Australasian* Medical Publishing Company, Limited—						
(i) Debentures: Series "A"-"D"	£17	5	0			
(ii) Debentures: Series "E"	£29	9	1			
	46	14	1			
				95	1	10
„ Excess of Expenditure over Income				61	9	10
				£4,971	9	2

We report that we have audited the accounts of the British Medical Association (Western Australian Branch) for the year ended December 31, 1952. In our opinion the accompanying Balance Sheet is properly drawn up and exhibits a true and correct view of the state of the Association's affairs as at December 31, 1952, and the attached Income and Expenditure Account is also properly drawn up and exhibits a true and correct view of the Association's affairs for the year. Both are in accord with the best of the information and explanations given to us, and as shown by the Books of the Association.

Perth,
Western Australia.

STOWE AND STOWE,
Chartered Accountants (Aust.),
Auditors.

REPORT OF THE LIBRARY COMMITTEE.

On behalf of the Library Committee, Dr. H. S. Lucraft, the chairman, presented the annual report. The report, which was adopted, is as follows.

We beg to submit to you the report of the committee on the Branch Library for the twelve months ended March 25, 1953.

It may be of interest to members, some of whom were perhaps not acquainted with the Library in its earlier days, to trace briefly its history over the past ten years or so and the progress it has made in that time.

The paucity of the Library's stock of books and periodicals in the past has not necessarily proved a stumbling block to its rendering an efficient service—rather, we think, it has been responsible for creating a situation whereby the Library has been forced to provide a service that has come to be appreciated, not only by members, but by individuals and departments far beyond the bounds of the Association.

The Library, of course, has not always been housed at the Royal Perth Hospital, although it had its origin there many years ago. From the Perth Hospital some time in the early 1930's it was transferred to "Chennell House", from whence in 1939 it was moved to "Shell House", and then in November, 1948, it transferred back again to its present quarters in the old part of the Royal Perth Hospital.

In 1943 the Branch had a membership of three hundred and twelve. At that time numerous members were on active service and some were prisoners of war, but nevertheless 740 requests for information were received and 1200 items borrowed in that year. Members of the visiting Allied Armed Services were making use of the Library and the Branch was made painfully aware of its inadequacies.

A Library Committee was appointed in 1944, but as no funds were available there was very little it could do. This state of affairs existed until 1948, when it was decided that some definite action would have to be taken with regard to the future of the Library, and with this in mind a report was prepared and submitted to Council. In the report the committee suggested various methods of raising the necessary finance, but none was considered satisfactory, and it was finally decided to approach the Medical Board for an annual grant.

The year 1949 found the Library settling in to its new premises at the Royal Perth Hospital. For the first time in its existence it was in receipt of a grant—a regular amount—from the Medical Board. For the first time, too, the report for the annual general meeting that year disclosed a decrease instead of an increase in the figures for the use being made of the Library. This was due entirely to the changed location, and once members accustomed themselves to combining their visits to the Library with their hospital days, and the resident medical officers became aware of its proximity, its recovery was complete. The Library Committee was now meeting once a month.

In 1950 the figures for visitors and borrowing again increased and surpassed those for any previous year. Many more members as well as ancillary medical services,

university professors, lecturers and honour students, officers of the Commonwealth Scientific and Industrial Research Organization and government departments *et cetera* were now making use of the Library's greatly enlarged stock of books and periodicals as well as its borrowing facilities. Arrangements were made for members to have access to the Library key after hours and on holidays. A delivery box was installed outside the Library door to enable members to call for and return material after hours, and this very quickly proved to be a valuable innovation.

About this time a member presented a number of photographic prints to the Branch to be sold for the purpose of raising funds for the Library. Many of these were sold, realizing a sum of £155 15s. 6d., but a few still remain and are available in the Library if any member is interested.

By 1951 the Library was regularly in receipt of approximately 130 periodicals. An historical section known as the "W. Horner Nelson Historical Section" and financed from a bequest by the late Dr. W. Horner Nelson was formed. In view of the greatly increased amount of borrowing from libraries in the eastern States and increased postal charges, the need for a microfilm reader was again stressed. A library book-plate, to an architect's design, was obtained and is being affixed to the inside front cover of all new books as they are received. In time it will be included in the older books and all bound volumes of periodicals. During this year the idea of selecting important references to articles in current periodical literature and circulating them each month together with details of new books and other items of interest in the form of "Library Notes" was introduced. The need for a printed library catalogue was appreciated, but consideration was deferred owing to lack of finance.

The number of periodicals being received increased to approximately 140 during 1952. Two thousand six hundred and ten requests for information were received, and 680 books and 2400 periodicals, microfilms *et cetera* were borrowed. A microfilm reader was obtained and a set of "Library Rules" drawn up. The rules contained notably few penalties as the committee was of the opinion that a policy of friendly cooperation would achieve better results than one of intimidation.

The year just concluded shows increases again over and above those for any previous year. Approximately 3000 requests for information were received. This figure represents 2570 actual visits to the Library and 430 requests received by telephone and letter. Eight hundred books were borrowed as well as 2850 periodicals. These figures do not include microfilms.

In spite of the very greatly enlarged stock of books and periodicals the amount of borrowing from libraries in the eastern States increased enormously. For example, in the twelve months just concluded eighty-four letters were written to the Barr Smith Library, University of Adelaide. These represented requests for seven books, 59 periodicals and 78 microfilms. In addition, material was borrowed from other libraries in South Australia, from the British Medical Association (Victorian Branch) and the Royal Australasian College of Surgeons and the University of Melbourne and the New South Wales Branch of the British Medical Association and the University of Sydney as well as our own Public, University and Departmental Libraries. Not to forget our own private members.

One hundred and five books have been added during the year and the Library now receives regularly approximately 145 periodicals. One hundred and fifty-six volumes were bound at a total cost of £237 12s. 3d., but unless additional funds are forthcoming the number of volumes bound for the period 1953-1954 will have to be considerably less. This will be a retrograde step as, although all periodicals received do not have to be bound, the majority of those that do contain two, and in some instances as many as three and four volumes per year. To reduce the amount of binding done, therefore, would mean that more and more material would accumulate, and already some quite large sets of periodicals are waiting to be bound.

The Library's entries for Pitt's "Union Catalogue of Scientific and Technical Periodicals in Australian Libraries" have recently been revised and forwarded to the Commonwealth Scientific and Industrial Research Organization in Melbourne so that when the next supplement to the catalogue is published it will contain the Library's complete periodical holdings to February, 1953.

Once again the committee acknowledges with very sincere thanks its indebtedness to the Medical Board. It is quite impossible to imagine how the Library could function without the grant it receives from the Medical Board. For various reasons the grant for 1952 was reduced, and towards the end of the year when the time came to renew the periodical subscriptions for 1953 the committee was faced with the prospect of having to cancel a large number of subscriptions owing to insufficient funds. In view of the fact that the Library was only just emerging from one period of darkness it was agreed that it would be disastrous at this stage to plunge it into another by discontinuing much-sought-after sets of periodicals, and so it was with this in mind that an appeal was made to members, mainly through the various specialist groups, to assist the committee over a difficult financial period.

The response from individual members and the majority of the specialist groups was most generous. Practically the entire number of subscriptions were renewed, but altogether during the year the sum of £885 was spent on books, periodicals and binding. This does not include the subscriptions that are being paid by private members, and there are still several very important periodicals missing from the Library's list—*Surgical Clinics of North America*, to mention only one.

It would seem then that the minimum amount required to maintain the Library at its present level of efficiency, exclusive of salaries, telephones, postages and other general expenses, is somewhere in the region of £950 to £1000 *per annum*. Of this £1000 the Medical Board provides £750.

During the year that has just passed the committee has been compelled to draw on the Library's Donation Account to the extent of over £200, and this fund has now been exhausted. It will be remembered that this particular fund originated in a spontaneous outburst of generosity among those present at a British Medical Association dinner several years ago. A recrudescence of the same spirit leading to a resurrection of the Library Donations Account would be very warmly welcomed by the members of your committee. It is obvious that, if the present standards are to be maintained, the Library will need additional support from some quarter.

Our sincere thanks are tendered to all those members who are assisting by bearing the cost of one or more periodicals. We would like to record also our thanks to Dr. E. R. Beech, the Secretary of the Post-Graduate Committee, who obtained from an anonymous donor the sum of £50 for the purchase of special equipment required for the Library's periodical accessions when the Post-Graduate Committee was unable to assist in this matter.

In May we received with very real regret the resignation of the Assistant Librarian, Mrs. Sinclair, formerly Miss Ruth Gee.

In July Miss Dallas Walsh was appointed as junior typist. Miss Walsh has quickly settled down and taken on the responsibility of her duties in a most efficient manner.

In the earlier part of this report some reference was made to the past history of the Library and to the manner in which it has grown from a very small nucleus of a few books and periodicals to its present state. The history of the British Medical Association Library in Western Australia is really the history of nineteen years' efficient and devoted service on the part of our chief librarian, Miss M. Bryan (Mrs. Button). It is difficult to record, with any approach to adequacy, the debt of gratitude we owe to Miss Bryan. Members have seen the gradual expansion of the Library facilities; the provision of suitable and comfortable furnishings; the installation of an outside box where books can be collected and returned after hours; the development of "Library Notes" giving details each month of items of interest in the most recent literature. The arrangement whereby the Library key can always be obtained by members, in cases of urgency, after hours, the provision of a suitable book-plate which is gradually being fixed to all Library material, and innumerable other details, the sum total of which adds up to the very efficient service, under comfortable conditions, which is available today to members of the Western Australian Branch of the British Medical Association—all of these ideas and many which I have not mentioned originated from and were put into effect by our librarian, Miss Bryan.

No doubt most, if not all, members present tonight make use of the Library from time to time, and all are fully aware not only of the efficiency with which their requests for information and assistance are invariably met, but also of the pleasant and courteous manner in which they are met. During all the nineteen years in which Miss Bryan has served the Library, she has always given the impression that she regarded it as a privilege to assist the members and not as a mere duty. There must be few libraries in which members have received such loyal, faithful and

efficient service as have members of the Western Australian Branch of the British Medical Association during the past nineteen years. During recent years, the Medical Board has made available the money which enabled the Library to expand to its present size. The conversion of that money into the very efficient library service of which we are so proud has been due largely, indeed almost entirely, to the work of Miss Bryan.

It is with the very greatest regret, therefore, that we have to announce the forthcoming resignation of Miss Bryan, who will be severing her connexion with the Library in May next. Her work, however, will continue to benefit us. She has placed the Library on a sound basis and your committee is confident that she will carry with her for all time the gratitude and good wishes of every member of the Branch for the invaluable service she has rendered.

We have been fortunate in obtaining the services of Miss Sheila Hagan, who will commence her duties as librarian early in April, and who for the first month or so will have the benefit of Miss Bryan's advice and assistance. We hope and believe that Miss Hagan will continue in the path blazed by Miss Bryan and that the same happy relationship between librarian and members will exist in the future as in the past.

In conclusion, your committee would like to appeal for the continued support of members. The Library needs all the help it can obtain, and the committee, at the risk of being wearisome, reiterates its urgent desire that by some means or other the Library Donations Account should be kept in existence. Any donation, no matter how small, will be gratefully received and acknowledged. Members who can contribute now will be thanked forthwith. Those who leave us a bequest in their wills will be remembered with gratitude. Those who do both will be placed in a special category and will have, in addition, the satisfaction of knowing that they have helped in a very worthy cause in a practical manner calculated to assist not only their fellow members but also the public in general. Surely no real doctor could lightly pass by an opportunity of this kind.

PRESIDENT'S ADDRESS.

The retiring President, Dr. John Day, then read his President's address (see page 833). A vote of thanks to Dr. Day for his address was carried on the motion of Dr. J. G. Hislop.

INDUCTION OF PRESIDENT.

Dr. John Day then inducted Dr. A. L. Dawkins as President for the ensuing twelve months. Dr. Dawkins thanked the members for his election.

VOTE OF THANKS.

A vote of thanks to members of the Council and to the office-bearers was carried on the motion of Dr. R. H. Crisp.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE HYGIENE OF THE VOLUNTEER UNIFORM.[1]

[*Australian Medical Gazette*, November, 1870.]

To the Editor.

Sir,

Now that the war panic is strong upon us, and that new uniforms are to be issued to the volunteers, it may not be out of season to say a word or two on the hygiene of the volunteer uniform. By the last mail we are informed that the London police are being supplied with a new helmet after the Prussian model with a large peak in front so as not only to shade the eyes but also to protect the nose (a matter of much greater importance than is generally imagined). There is also a peak behind to prevent rain from entering between the neck and the collar coat.

We have also borrowed from Prussia. But it is the Prussian mütze or skull cap called a foraging cap, a most unscientific and absurd head dress. It allows the whole forehead, nose and eyes to be exposed to the glare of the sun and to all weathers. The forehead and nose become blistered and the eyes bloodshot even on a single review day. What would be the case if men were really to meet an enemy in such a state? The men would fight certainly, but at a great disadvantage. And again if the men had to undergo great fatigue in marching, in a hot sun, there would, to a certainty, be a large amount of insolation. No manner of head dress could be devised that would induce such effect more certainly than the present volunteer cap. Then again as to the "jumper". Shortly after the Crimean War, the British Government abolished the tail coat on account of its unhealthiness and the tunic became the dress, for all line regiments which were dressed in tunics, suffered less from diarrhœa and dysentery than those which had not the skirt of the tunic to protect the stomach and internal organs from the wet and the vicissitudes of the climate. Hundreds of lives were thus unnecessarily sacrificed; and thousands of lives before the old regulation leather stock was done away with. Do not let us be so stubborn; for though SARTORIUS is a great god—very nearly, if not quite, as great as MARS in modern military matters—let us, in common sense, worship poor HYGEIA a little also.

If a jumper we must have, why not have one like the fatigue dress of the regulars. It would cost no more than the present Garibaldeon jumper, and I think the sartorial mind will acknowledge that it may be made equally showy, if they wish it so. The shoulder belt, too, though not to the same extent as the cross belt, is a source of what is called the soldier's *callus*, a hard corny substance produced on the chest by the pressure of the belt, and according to all military surgical authority, a great source of heart disease in the army. Our volunteers have not found it out yet with their half-dozen rounds of blank cartridges, but let them have their full complement of ball cartridge in their cartridge boxes and a long march; they will find out how oppressive to the breathing is the belt across the chest.

The waist belt again is too wide and too clumsy. It should be made so that it is easily tightened or slackened. Man instinctively, when he is going into violent action, tightens his waist. The officer going into action tightens his sword belt. The naked savage puts a string round his waist. By so doing he assists the abdominal muscles to support the liver and other internal viscera and prevent them from jolting, and the scriptual idiom of "girding up the loins to battle" shows that it was well known to the ancient Jews. After violent exertion it is a great relief to again loosen the belt; therefore it should be easily done.

I am, sir, your obedient servant,

Vox.

[1] From the original in the Mitchell Library, Sydney.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 26, of May 7, 1953.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

To be Temporary Major, 31st March, 1953.—1/8053 Captain A. J. Splatt.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

VX700320 Captain (provisionally) D. N. Hawkins relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the honorary rank of Captain, 19th March, 1953.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 1/39109 G. J. McCafferty and 1/39133 C. L. Cilento. 1/39133 Captain C. L. Cilento is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District), 27th March, 1953. To be Captain (provisionally), 10th March, 1953: 1/66025 Robert Molesworth Goodwin.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/111489 Captain P. J. Robinson is appointed from the Reserve of Officers, 20th February, 1953. To be Captain (provisionally), 2nd April, 1953: 3/101819 James Herbert Stephens Martin.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—To be Temporary Lieutenant-Colonels: Majors 4/35217 F. B. Turner, 26th February, 1953, and 4/32032 J. R. Barbour, 2nd March, 1953. To be Major, 8th April, 1953: 4/31907 Captain (Temporary Major) R. A. Burston.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/32055 Major L. W. Martin is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (5th Military District), 21st April, 1953. 5/26509 Captain (provisionally) H. J. H. Colebatch relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (5th Military District) in the honorary rank of Captain, 25th March, 1953. To be Temporary Major, 21st April, 1953: 5/21508 Captain (Honorary Major) M. G. F. Donnan.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 6/5128 D. B. Nathan and 6/9210 A. C. D. Corney. 6/15409 Captain (provisionally) J. M. L. Hunn relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District) in the honorary rank of Captain, 3rd March, 1953. To be Temporary Major, 31st March, 1953: 6/5128 Captain D. B. Nathan.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—To be Honorary Captains: David Adam Thatcher, 3rd April, 1953, and Peter Browne Rowland, 13th April, 1953.

2nd Military District.—To be Honorary Captains: Philip Arthur McSharry Powell, 9th March, 1953; John Vincent Carlin Brassil, 10th March, 1953; Peter James Miles, 15th March, 1953; and Peter Bethune Buchanan, 27th April, 1953.

ROYAL AUSTRALIAN AIR FORCE.

Air Force Reserve: Medical Branch,

The following former officers are appointed to commissions with the rank of Flight Lieutenant: J. P. Fleming (263942), 15th November, 1952, K. M. Twiddy (419145), 27th January, 1953.

The following are appointed to commissions with rank as indicated: (Flight Lieutenant (Temporary Squadron Leader)) Ralph Jackson Cato (297539), 18th December, 1952; (Flight Lieutenant) Bruce Stephen Mackie (268006), 28th October, 1952, Atholl Lovat Frazer (297536), 13th November, 1952, James Richard Henry Watson (297537), Charles Lyall Stewart (455272), 19th November, 1952, Kevin James Fagan (268007), 24th November, 1952.

Obituary.

STUART GALLOWAY GIBSON.

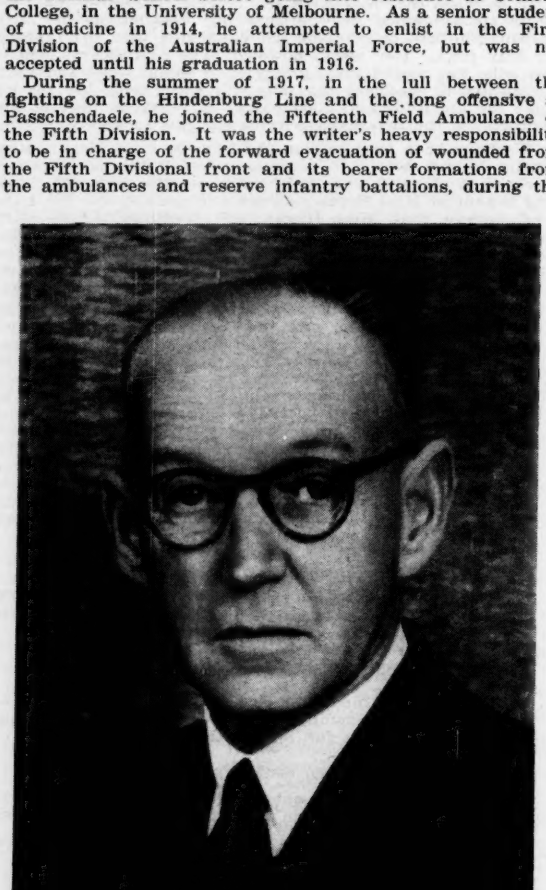
WE are indebted to Dr. W. E. L. H. Crowther for the following appreciation of the late Dr. Stuart Galloway Gibson.

Stuart Galloway Gibson died unexpectedly at Hobart on March 23, 1953, of coronary occlusion secondary to an operation for gall-stones performed three days before. He was a Tasmanian of the fourth generation, his great-grandfather, David Gibson, having come to the colony in the very early days of its settlement. The pastoral properties of his descendants and their famous Belle Vue stud of merino sheep are well known in Australia and beyond.

Stuart's father, George Harry Gibson, broke away from the family tradition to enter the University of Edinburgh from which he graduated M.B., Ch.M. in 1887. Establishing himself in practice in Hobart, he was for many years senior physician to the Homœopathic Hospital and continued his association when it was taken over by the Church of England and renamed Saint John's. A brass plaque in the main hall commemorates his long professional association with the hospital. He lived with his large family of five sons and four daughters above us in Macquarie Street, in the house where my grandfather had practised for over forty years and which is now the house of the headmaster of the Hutchins School.

Stuart, the third son, with his brothers, was educated at the Friends' School before going into residence at Ormond College, in the University of Melbourne. As a senior student of medicine in 1914, he attempted to enlist in the First Division of the Australian Imperial Force, but was not accepted until his graduation in 1916.

During the summer of 1917, in the lull between the fighting on the Hindenburg Line and the long offensive at Passchendaele, he joined the Fifteenth Field Ambulance of the Fifth Division. It was the writer's heavy responsibility to be in charge of the forward evacuation of wounded from the Fifth Divisional front and its bearer formations from the ambulances and reserve infantry battalions, during the



Polygon Wood phase of the September fighting. Gibson, then a bearer captain, with men from his own unit was stationed at a wagon loading post just below Hooge Crater and was awarded the Military Cross for his courage and example in disposing of the many casualties from this heavily shelled position. He remained with the Division for part of the time as a regimental medical officer, until after the armistice.

On returning to Tasmania, Gibson commenced practice at Moonah near Hobart and very shortly after married Leonora, daughter of Major Whitham of the Indian Army. The death of Dr. Gibson, senior, in 1925 brought about a return to the old home in Macquarie Street and his taking over of the family practice; here he remained until his last illness. As with so many of his period, active service had absorbed the years that otherwise might have been given to house appointments and preparation for specialization. In spite of this grave handicap Gibson became an outstanding general practitioner, as well as honorary physician to the Children's Hospital. Indeed, for the last decade and more he had been recognized as a very able anæsthetist, being the first in Hobart to use a McKesson portable machine, which he took from hospital to hospital as required. He might well have specialized in this art, but the bonds with

his older patients and ex-servicemen were too firm to be broken.

For some thirty years Gibson was on the active list of the Commonwealth Military Forces and was one of a considerable group who regarded their period of military training at the annual camp at Mona Vale as an annual reunion to be attended and enjoyed. He reached the rank of lieutenant-colonel, with command of the Twelfth Field Ambulance and award of the Efficiency Decoration. Mobilized in 1939, he was commanding officer of the 111th Australian General Hospital at Campbell Town until late in the war, when he developed symptoms then regarded as due to a duodenal ulcer. As a result he was "boarded" and placed on the retired list.

Mention must be made also of the long years of service to the Tasmanian Branch of the British Medical Association, as a member of Council and as President for 1950. He was, again, one of the two Tasmanian representatives on the Federal Council from 1934 to 1940.

All these interests told on his strength, and a little over two years ago he was operated upon for Buerger's disease. At the end of last year, with the graduation of his son, Dr. David Gibson, it seemed that he and his wife might at last take a long holiday, join their daughters in England and attend the Coronation. Plans had been completed to leave this month (April) by the *Orion*, but an exacerbation of his condition brought about his entry to hospital and its sad sequel.

Stuart Gibson had been from boyhood of slight physique and fair complexion, and in consequence gave the impression of being younger than his years. He was precise in manner and walk, and it was his habit to dress with care and taste. In manner he had a certain reserve and reticence in speech. These qualities were balanced by a dry humour, quiet geniality, self-reliance and loyalty which gained him the lasting affection of his patients and colleagues.

He and his wife were fortunate and exceptionally happy in their family life. With their two daughters and son they made their home always "open house" to visitors and friends, especially the young. It was their custom to take their recreation together at the Royal Hobart Golf Club (for which Gibson played "A" grade and served on the executive) where they played on one or two afternoons a week; or as the weather hardened to fill the two cars with their friends for a hurried visit to National Park for winter sports. Gibson was very competent on skis and found an immense pleasure in the success of his daughter and son, Meg and David, in such competitions.

The very large part Stuart Gibson had taken in the life of the community in which he worked was shown by the gathering at his funeral on March 25. On this occasion the Dean of Saint David's Cathedral with deep sincerity spoke of him as "a faithful servant of God and a good companion". His friends and old patients who were present felt this to be a most fitting tribute.

Honours.

CORONATION HONOURS.

THE following Coronation Honours have been awarded by Her Majesty Queen Elizabeth II.

Dr. Gilbert Brown, of Adelaide, has been made a Commander of the Most Excellent Order of the British Empire.

Dr. Kenneth Barron Fraser, of Brisbane, has been made a Commander of the Most Excellent Order of the British Empire (Military Division).

Dr. Francis John Graham, of Sydney, has been made a Member of the Most Excellent Order of the British Empire.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Churchward, Sydney Lewis, M.B., B.S., 1951 (Univ. Sydney), 51 Victoria Street, Burwood, New South Wales.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 9, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	1(1)	2(1)	..	..	..	..	..	..	3
Amœbiasis	..	1(1)	..	..	..	..	..	..	1
Ancylostomiasis	..	..	..	..	1(1)	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	2(2)	..	..	..	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1(1)	3(3)	18(11)	..	..	..	..	..	22
Diphtheria	14(10)	8(6)	5(3)	1	7(6)	1(1)	..	..	36
Dysentery (Bacillary)	..	..	1(1)	47(7)	..	..	..	..	48
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	9(4)	..	..	6(3)	..	..	..	15
Lead Poisoning	..	..	..	..	2	..	..	..	2
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	2(1)	..	..	1	..	..	..	3
Ophthalmia	..	..	..	..	26	..	..	..	26
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	12(9)	10(2)	6(1)	12(9)	1(1)	2	..	1	44
Puerperal Fever	..	1	..	..	..	..	..	..	1
Rubella	..	26(9)	..	..	8(2)	..	..	..	34
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	5(4)	116(63)	1	5(5)	1(1)	2	..	..	130
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1(1)	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(22)	43(32)	25(20)	8(6)	13(10)	1	..	1	123
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.

Medical Practice.

POLICE OFFENCES (AMENDMENT) ACT, 1908, AS AMENDED.

THE following proclamation, published in the *New South Wales Government Gazette* of June 5, 1953, is published at the request of the Under Secretary to the New South Wales Chief Secretary's Department.

I, Sir John Northcott, Knight Commander of the Most Distinguished Order of Saint Michael and Saint George, Companion of the Most Honourable Order of the Bath, Member of the Royal Victorian Order, Lieutenant-General on the Retired List of the Australian Military Forces, Governor of the State of New South Wales and its Dependencies in the Commonwealth of Australia, with the advice of the Executive Council, do, by this my Proclamation, declare that Part VI of the Police Offences (Amendment) Act, 1908, as amended, shall apply to:

> Methorphan (3-Methoxy-N-Methylmorphinan), its salts and any preparation, admixture, extract or other substance containing not less than one-fifth per centum of Methorphan

in the same manner as it applies to the drugs mentioned in paragraph (*a*) of subsection (2) of Section 18 of the said Act.

Signed and sealed at Sydney this twentieth day of May, one thousand nine hundred and fifty-three.

By His Excellency's Command. C. A. KELLY.

Medical Appointments.

Dr. W. J. R. Wyness has been appointed honorary clinical assistant to the X-Ray Department of the Royal Adelaide Hospital.

Dr. R. J. D. Turnbull has been appointed a member and Vice-President of the National Fitness Council of Tasmania.

Dr. F. J. Scanlan has been appointed deputy medical superintendent in the Division of Mental Hygiene of New South Wales.

Dr. J. N. Main has been appointed medical superintendent in the Division of Mental Hygiene of New South Wales.

Dr. R. J. Farnbach has been appointed a member of the Milk Pasteurization Committee in the Department of Agriculture of Victoria.

Dr. A. W. Riseborough has been appointed a public vaccinator for the Shire of Minhamite, Victoria.

Dr. R. A. Brown has been appointed medical officer for Tuberculosis Services at the Royal Adelaide Hospital.

Dr. E. V. Knight has been appointed medical officer, State Government Insurance Office, Brisbane.

Professor E. S. J. King has been appointed deputy chairman of the Advisory Committee to the Mental Hygiene Authority of Victoria.

Australian Medical Board Proceedings.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners: Kenihan, Robert Austin, M.B., B.S., 1916 (Univ. Adelaide); Jones, Bruce Patrick, M.B., B.S., 1950 (Univ. Melbourne); Thomson, William McLaren, M.B., B.S., 1947 (Univ. London).

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners: Hawke, Pamela Joan, M.B., B.S., 1953 (Univ. Sydney); Donnelly, Michael Francis, M.B., B.Ch., 1944, M.D., 1949 (National Univ. Ireland), M.R.C.P. (London), 1949; Ashworth, Eveleen, M.B., B.S., 1924 (Univ. London).

The following additional qualification has been registered: Lavers, Kenneth Wilcox, D.C.H., R.C.P. and S. (England), 1951.

Deaths.

THE following deaths have been announced:

ALLESTER.—Edwin Marston Allester, on May 30, 1953, at Huonville, Tasmania.

GURNEY.—Guy Stephen Gurney, on June 3, 1953, at Bowral, New South Wales.

HARDY.—Lowen Alexander Hardy, on May 27, 1953, at London, England.

JUETT.—Alexander Juett, on May 19, 1953, at West Perth, Western Australia.

Diary for the Month.

JUNE 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 17.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 18.—South Australian Branch, B.M.A.: Clinical Meeting.
JUNE 18.—Victorian Branch, B.M.A.: Executive of Branch Council.
JUNE 23.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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